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COMMON CARDIAC PROBLEMS: SYNCOPE, CARDIAC NEUROSES, PRÆCORDIAL PAIN.[1]

By M. D. SILBERBERG, M.D. (Melbourne),
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Syncope.

SUDDEN events affecting the integrity of the brain and heart necessarily strike at the foundations of life itself and are naturally enough associated with grave concern and apprehension.

Such is the state of affairs induced by syncope of whatever type or mechanism. It is therefore not strange that the patients and their relatives are highly perturbed by such an occurrance as a fainting attack. The aspect of deathly pallor, of dilated pupils, cold sweat, slow sighing respirations is sufficiently alarming, not to mention the slow feeble heart action and pulse with low blood pressure. While it is true that most patients soon recover and, apart from the feeling of limpness and some prostration for a short interval, are none the worse, yet there is the unpleasant knowledge that similar attacks have proved fatal. It is this that contributes to the anxiety in both patients, relatives and physicians.

How are we to distinguish between the grave serious forms of syncope and the harmless varieties?

We find by experience that the innocent forms are dependent on a disturbance of nervous mechanism—a disharmony of balance of the autonomic system between the sympathetic and parasympathetic distributions—an overaction of one or other part of the important involuntary nervous system. The disturbance acts through the vagal mechanism and vasomotor systems and produces its effects by circulatory depletion of the higher centres, along with anæmia of the brain and medulla.

This can be brought about in diverse ways: (i) either by overaction of the vagus and consequently vagal inhibition, (ii) vasomotor pooling in the large abdominal vessels or (iii) at times a depressor vagal mechanism induced by a sudden rise of pressure in the aorta.

The commonest variety is by vagal inhibition. This may be induced by many afferent stimuli through distance receptors, aural or more usually visual impressions or by exteroceptors or interoceptors. In the susceptible, various emotional causes bring it into play; the commonest is perhaps fear, for example, the sight of blood, a medical student's first visit to the operating theatre, a visit to the dentist, taking samples of blood for diagnostic purposes are common enough examples, especially in young people and children.

The shock of bad news, more rarely of joy may bring the mechanism into action. Of exteroceptors pain is a potent cause and under some circumstances gastric distension may prove so.

Vasomotor causes of syncope are fairly common, as when occurring after a hot bath or in a hot air cabinet or in crowded stuffy public gatherings. The hypotonic visceroptotic shop girl standing for a long time and fatigued by a hot day illustrates not infrequently this failure of the vasomotor sympathetic tonicity.

The depressor mechanism protects against a dangerous rise of blood pressure, especially in the hyperpietic and is therefore more usual in people past middle age. The widespread distribution of the vagus system is well illustrated by the symptoms of an attack not occurring too suddenly.

The first thing noticed is an unpleasant sinking sensation about the epigastrium, salivation and a cold clammy sweat, very similar to impending seasickness. Then the more direct vagal circulatory effects appear from pulse slowing and low blood pressure, giving intense pallor, a feeling of chilliness or sweat, a limpness and loss of tone of the muscles and the patient sinks down more or less unconscious with slow sighing respirations, dilated pupils and possibly vomits or voids urine or fæces. The stage of recovery still shows some pallor and feelings of prostration and lack of desire for movement and perhaps sleepiness.

Some of the above effects are no doubt a result of overflow of impulses to the mid-brain and sacral parts of the parasympathetic. They clearly illustrate the widely spread diffuse effects of the parasympathetic contrasted with the localized accurate reflexes from the somatic nerve supply.

The type of patient who faints in a hot bath, usually possesses poor nervous, visceral and somatic tone, not infrequently an obese flabby middle aged or elderly person. In themselves the attacks, though alarming, are not serious and I have known of patients living for years in good health after such an event.

They have the advantage that the patient seeks an overhaul and the obesity and lack of tone can be improved. I have seen a number of obese flabby men with low blood pressure who have had sudden and severe syncopal attacks after a heavy meal.

A business man of fifty-three years, of obese flabby type with a blood pressure of 120/80 millimetres of mercury, falling to 110/80 millimetres on standing, had four severe attacks following on meals and flatulent distension. In one he fell heavily, knocking over a chair, making his nose bleed and bruising himself. His habits were temperate and he had been accepted for life insurance as first class. His physical endurance was good and he could play two rounds of golf without distress. His business life was strenuous and associated with much worry. Cardiac and general examinations and electrocardiograms revealed nothing of note. Evidently the attacks were due to a gastro-vagal mechanism.

In another apparently similar case it transpired that the syncope was due to a large duodenal hæmorrhage.

In a man of thirty-six years syncope was associated with large meals, excessive alcohol and tobacco, but curiously enough every attack was preceded by an aura of loud buzzing in the right ear. On two occasions his doctor gave him an emetic and brought away offensive vomitus

[1] Read at a meeting of the Victorian Branch of the British Medical Association on May 2, 1928.

and undigested food. No neurological or cardio-vascular abnormalities were found and the special senses were normal. Toxic epilepsy may have been the cause. He was told his mouth screwed over to the left. In another attack he had time to take out his false teeth and lie down and was unconscious for twenty-five minutes, he called out and his breathing was noisy and he vomited after each attack. The attacks have become less frequent since he has paid attention to dietetic and tobacco excesses.

It is difficult at times to differentiate between *petit mal* and syncope.

A nurse aged thirty-four years from a country hospital was referred for an opinion about her heart because of occurrence of faints. She stated she had had many attacks over a period of years, but they were worse in the last twelve months. She described them as starting with a burning sensation over the abdomen, which gradually spread; she was barely conscious, no struggling occurred; attacks were usually premenstrual and more likely with strain and emotion; there was no family history of epilepsy. A nurse colleague stated that she observed pallor, staring, speech thickened and that the tongue was bitten; there were movements about the chin and urine was voided. Examination revealed no abnormality and the opinion was given that this was a case of epilepsy.

A depressor type of attack was observed in a lady of seventy years, whom I had known well for years. She usually had a systolic blood pressure in the region of 200 millimetres of mercury; she had had several attacks of paroxysmal fibrillation, the heart being enlarged and the radial arteries showing calcified beading. On a hot humid day at a public gathering she felt faint; just as she managed to get outside the building she became unconscious and had convulsive movements of the face. She came round in a few minutes and had no recollection of the first aid procedures which had been adopted. She was none the worse for the experience, which was very alarming and looked like the onset of apoplexy. She lived for ten years after this, dying suddenly on arising from bed.

A violent blow over the heart in boxing or on the solar plexus may bring on syncope, no doubt through inhibition of vasomotor mechanisms.

Drowning following a heavy meal is probably brought about by depletion of the cerebral centres, by pooling in the splanchnic vessels or by a gastro-vagal reflex inhibition.

Low blood pressure is not unusually associated with syncope, especially in obese types and after meals with gastric distension. No doubt a further fall of the already low blood pressure is readily brought about by vagal inhibition or splanchnic pooling with resulting cerebral anæmia and unconsciousness. However, one has to be particularly careful in these types to eliminate myocardial lesions, such as fatty heart. A good exercise response—golf *et cetera*—is well tolerated without dyspnœa or pain; no crepitations at the bases and no distended veins in the neck, absence of œdema of the legs towards the end of the day all point to a more or less intact myocardium. The size of the heart may be difficult to assess without an orthodiascope and weak heart sounds may mean nothing more than a thick chest wall and emphysema.

A tall thin man of fifty-eight years, accustomed to some nine whiskies daily and with evidence of valvular degeneration, got out of bed at 2 a.m. to urinate and was soon after found unconscious. His doctor found the blood pressure 126 millimetres of mercury, the pulse rate 80 to 90, with some irregularity. After recovery the systolic blood pressure was 160 millimetres of mercury and the diastolic pressure 100 millimetres, the pulse was regular; evidently the change of posture and emptying of the bladder caused a sudden fall of blood pressure probably through splanchnic pooling.

Examples could be multiplied, but I would like to pass on to a consideration of a group with more grave prognosis dependent on cardiac disease.

The underlying cardiac pathology comprises fibro-fatty and atrophic changes, toxic and inflammatory conditions. The ætiology is necessarily varied, ranging through the effects of syphilis and rheumatic fever, chronic toxæmia, senile changes and arteriosclerosis, metabolic and endocrine disturbances *et cetera*. Such pathological change naturally is serious and often irreversible.

Cardiac irregularities often occur in this group, though on the other hand we have to confess we know very little of the pathological change causing, for example, extra-systoles and fibrillation. There is much to be said for the view that extra-systoles may arise from functional disturbances acting through the nervous mechanism and excited by stimuli arising in reflexigenous areas such as the gastro-duodenal regions.

I have seen several examples of syncope induced by a short paroxysmal series of extra-systoles arising in the ventricle.

The clearest instance was in a male, *ætatis* twenty-five years, ironmoulder, first seen in January, 1925. He complained of taking peculiar "turns" in the previous five weeks, as many as eight occurring in the hour. He noticed that on walking he lost control of his legs and had fallen; he might be momentarily unconscious for a few seconds and felt weak for about two hours afterwards. He had had two attacks of rheumatic fever when a child, there being no family history of epilepsy. On examination he was seen to be a tall man, of fair nutrition. The apex beat was 13·1 centimetres (five and one-quarter inches) from the mid-line. There was a slight mitral systolic bruit. The rhythm was very curious and showed frequent short bouts of tachycardia of four to six beats. The cardiograms showed these to be due to short paroxysms of ventricular tachycardia arising from the right ventricle. The "turns" complained of coincided with the tachycardia and it is possible to be sure that a long attack would be very dangerous and likely to produce fatal syncope. I have tried to follow up this patient, but my letters were unanswered.

Palpitation due to extra-systoles can frequently be identified from the patient's own graphic description. He may note the small beat as if the heart "flopped" or as if it "turned over" or the pause is more stressed with a sudden realization that the heart has missed a beat, followed by an unpleasant thump, as the heart, now overfilled, gives a strong beat transmitted to the chest wall.

Stokes Adams Attacks.

Syncope from Stokes Adams attacks in heart block is better known and there is no difficulty in recognizing the condition when, as usually, the ventricle rate is in the region of thirty per minute.

Two clinical signs are useful in the diagnosis of heart block. One is a recurring unduly loud first sound which is produced from reinforcement by an auricular systole coinciding with the ventricular systole; the other sign is that the auricular systolic sounds may be heard in the long pauses.

I have, however, seen several patients suffering from complete heart block with a ventricle rate as high as forty-eight per minute, but this is unusual.

Syncope is more likely to occur when two to one block is changing over to complete block. The ventricle has a latent period before it initiates its own rhythm and in this period syncope may occur from cessation of circulation. When complete dissociation of auricle from ventricle is established, patients may carry on for years without Stokes Adams attacks and with remarkably good efficiency.

Why should complete block patients suffer from syncope, that is, why should the ventricles every now and then cease to beat? Is it muscle exhaustion, nervous inhibition or ventricular fibrillation? I think there are various mechanisms.

In a syphilitic patient, a man aged fifty-eight years, under the care of Sir James Mackenzie and Sir Thomas Lewis, I took a continuous strip of polygraph tracing from the apex beat for twenty minutes, during which time he was having frequent syncopal attacks from asystole of various durations, ranging from a few seconds up to thirty seconds. He continued having the attacks for some twenty-four hours and died in one. Autopsy revealed extensive gummatous changes throughout the heart. The usual ventricle rate was thirty per minute, but preceding each asystolic period was a ventricular paroxysm of seventy per minute. At the cessation of the paroxysm there was a varying latent period before the ventricle resumed its beat; in this interval the unconsciousness occurred.

In another syphilitic patient with complete block there occurred every now and then ventricular paroxysmal tachycardia at two hundred per minute. It was the very low output of blood at this stage that produced syncope and not the usual cessation of systole.

It would not be difficult for such a patient to develope ventricular fibrillation which would of course prove fatal, unless it was of very brief duration.

In one case of complete block, definite gastric distension was invariable before syncope, which is very suggestive of gastro-vagal inhibition.

Dr. Ronald Small asked me to see a patient whose heart had undoubtedly ceased to beat. No pulse or heart sounds could be made out; there was no attempt at breathing; the pupils were dilated and the face pale. The relatives were lamenting distressfully and all seemed over. Dr. Small found some adrenalin in his bag, hastily made an intracardiac injection and the patient, a woman, aged sixty-eight years, recovered. It was naturally difficult to assess the time of cessation of the heart action, but it must have been about two minutes. For about five minutes after consciousness returned the patient was blind, but complete recovery of all faculties occurred, though there was no memory at all of the events. After this she had a number of attacks in which intracardiac and subcutaneous injections of adrenalin were again used, but she eventually succumbed forty-eight hours after the above incident.

Although one may succeed in bringing a patient out of prolonged syncope by intracardiac injections of adrenalin (and it is obvious that no other route will be efficient because there is no circulation) the pathological cause is not removed and therefore death may close the scene. However, let me again emphasize that complete heart block is compatible with many years of useful life and may not be associated with syncope.

Patients with fibroid and fatty myocardia may have syncopal attacks and in these a fatal termination may readily occur, especially following some physical effort or powerful emotional excitement.

Why do men and less often women fatally collapse on the golf links or at public gatherings?

The mechanism is most likely ventricular fibrillation, the cause being effort or excitement; the pathological basis is varied. The same applies to fatal angina. Chloroform deaths in the early stage of anæsthesia are almost certainly due to ventricular fibrillation, though the late Dr. Embley favoured vagal inhibition and vasomotor failure. How can such an event be recognized? The signs are: abrupt abolition of the pulse and heart sounds, sudden fall of blood pressure, unconsciousness, dilatation of the pupils, intense pallor followed by lividity or cyanosis. The muscular relaxation may be preceded by a brief phase of rigidity and there is a continuance of slow sighing respirations after the pulse ceases. If one is on the spot, as at operations, prompt action is essential, an intracardiac injection of adrenalin should be immediately given and if unsuccessful in restoring the beat, heart massage should be attempted. Definite recoveries have been recorded by such means; it is a time when desperate disorders need desperate remedies.

Ventricular fibrillation necessarily arrests the circulation and is incompatible with life unless of very brief duration.

With auricular fibrillation and the auricle out of action as a regularly contracting reservoir, blood still gets to the ventricle because of venous pressure and is pumped out in the usual way and, although the ventricle is stimulated to contraction irregularly and unevenly, it may do so with reasonable efficiency for years.

Occasionally fatal sudden syncope occurs in auricular fibrillation and it is here likely that ventricular fibrillation is the mechanism producing death.

In auricular flutter the auricle is beating at a very high speed, for example, 260 per minute, and usually the conduction system conveys only every second stimulus, so that the ventricle will maintain a steady one hundred and thirty per minute. On rare occasions all the auricular stimuli traverse the bundle of His and the resulting ventricular output at, for example, 260 per minute, will be very small and unconsciousness may occur. Mackenzie held the view that prolonged unconsciousness ending in recovery is sometimes due to this mechanism, because so long as even a flicker of circulation is maintained, the tissues are kept alive.

In sudden deaths from angina it is highly probable that the actual mode of death is ventricular fibrillation, whatever the underlying pathological change may be.

The treatment of simple syncopal attacks is largely a matter of first-aid measures: loosening the clothes, keeping the head low, giving diffusible stimulants *et cetera*. For vasovagal attacks an injection of atropine with or without strychnine is useful.

Cardiac Neuroses.

The next problem, frequently encountered in cardiology, is that of so-called cardiac neuroses. We have not yet evolved an apt name to embrace this condition. You will recognize the type under such terms as irritable heart, vasomotor asthenia, cardiac neurasthenia, neuro-circulatory asthenia, disordered action of the heart and effort syndrome.

The symptoms include tremulousness, sweating, nervous instability, palpitation and breathlessness which are all readily aggravated by minor degrees of effort or excitement. Quite often giddiness, feelings of faintness and stabbing præcordial pain are prominent, as is also pronounced fatigability, all leading to striking subefficiency for manual occupations.

If, as is not infrequent, the symptoms show up after some incident of strain and a bruit is heard, a diagnosis of strained heart is commonly made and forms the basis for many a claim under the *Workers' Compensation Act*.

It is necessary to realize that this is a common disorder, frequently misinterpreted. It is not a primary heart disease, but rather a disturbance of the sympathetic nervous system in which the heart shares. The disturbance is based on toxic states, anxiety conditions and inherent or acquired nervous instability. Strain is sometimes a factor, but too often it is the associated anxiety neurosis, born of ideas of "strained heart" and all the dread which this implies to the lay mind and which keeps up the condition. All too frequently it becomes a case of a "doctor-made neurasthenic" should a diagnosis of "strained heart" or "leaking valve" be loosely made.

It was because of the fear aroused by a diagnosis implying heart disease, that terms referring to "heart" or "cardiac" were discarded. Hence the use of such a cumbersome label as "neuro-circulatory asthenia," though this is descriptive enough. Lewis introduced the phrase "effort syndrome" because it was non-committal as regards ætiology and pathology, but still more because the symptoms are exactly those produced by strenuous effort in the healthy individual, the difference being that the symptoms are now manifest with minor degrees of effort.

The syndrome is also similar to that seen in chronic toxic conditions, for example, early tuberculosis, thyreotoxicosis *et cetera*. It is very necessary to eliminate these and other forms of organic disease before a diagnosis of functional disease is made. It is a mistake to think that only neurasthenics or persons of nervous instability acquire effort syndrome states. It occurs in all types and in men, women and children.

It is natural enough that those with poor physique, poor visceral and vasomotor tone, often visceroptotic and hypotonic, readily break down under conditions of stress and strain, sustained anxiety, toxæmia from chronic focal infections or after acute infections. This group is a prominent one, but burly, placid individuals may also exhibit the syndrome.

In women it is common following on many weeks or may be months of nursing a sick relative, with its attendant anxiety, mental strain, broken sleep, irregular meals and fatigue. An intercurrent infection like influenza, tonsillitis or pneumonia *et cetera* on top of such fatiguing conditions often produces this irritability of the sympathetic system in which tachycardia is a prominent symptom. All grades of the condition are observed; mild cases clear up easily, moderate cases take some months for improvement and chronic cases go on indefinitely. I see numbers of returned soldier patients who have had effort-syndrome symptoms unchanged for eight to twelve years.

The prognosis as regards life is good, but for economic efficiency very poor. This latter does not apply to sedentary workers so much as for occupations needing physical exertion. A very few patients out of some hundreds seen by me have developed paroxysmal tachycardia and auricular fibrillation.

The heart does not enlarge or degenerate to a degree in any way comparable to that seen in thyreotoxic conditions. Latent or non-progressive tuberculosis is a toxic basis for a considerable proportion.

The basal metabolic rate has not been investigated in a sufficient number of patients. Hyperthyreoidism has often been blamed as the underlying cause. Certainly the symptoms are similar, but in effort syndrome types the thyreoid does not enlarge over a period of years, exophthalmos does not occur, nor is the heart so gravely affected. Sympathetic disturbance is the rule in thyreoid intoxication and it is well recognized that anxiety conditions and toxic states may bring on thyreotoxicosis. Hyperthyreoidism as the underlying cause is thus more apparent than real.

If the basal metabolic rate is high and there is no sign of an enlarged thyreoid, is thyreoidectomy justified? An expression of opinion on this point would be welcomed, particularly in regard to end results. In the chronic cases medical treatment is most unsatisfactory. During the war the authorities recommended graduated exercises. This did good in the mild cases, but failed in the severer ones.

Treatment should be directed to allaying the anxiety neurosis, first by a simple explanation of the nature of the condition: that it is not a primary heart disease, but rather a disturbance of the nervous controls. Avoid a diagnosis of "strained heart" or "leaking valve" unless a careful non-technical explanation of the real significance is given at the same time. Stress the fact that there is no danger to life, though it may mean subefficiency for manual work. This presupposes elimination by careful examination of serious organic disease. Endeavour to adjust other causes of fatigue and anxiety neurosis. These may be domestic, financial, economic, sexual, uncongenial work, long hours with too much responsibility *et*

cetera and may be beyond the scope of the medical practitioner to remedy. However, good advice at least can be given and the importance of psychical harmony stressed.

In those with the neurotic visceroptotic hypotonic diathesis one may be truly "up against it." Improvement of tone should be aimed at, but often these patients are seen too late. They should be recognized in childhood and suitably instructed—no easy problem. One is "up against" faults in heredity and what is perhaps more important, faults in upbringing and environment. Added to this is low resistance to catarrhs, acute and chronic infections and to tuberculosis. It is a big problem and may be insoluble. Of drugs the bromides are the most useful and glycero-phosphates and calcium salts do some good in spite of their alleged non-absorbability. Strychnine makes the already over-excitable nervous system worse. Gland preparations are as a rule useless. Digitalis and other heart drugs are commonly prescribed, but in my opinion are quite valueless. We need a safe sedative for the unruly sympathetic and, as far as I know, we have not got one.

The vexed question of the relation to focal infections usually arises. In my experience attention to sources of infection does good in early cases; in late cases the results are disappointing. In all cases the ordeal of operation, whether on teeth, tonsils, antra, gall bladder *et cetera* usually causes a temporary exacerbation of symptoms and it is well to warn patients of this. It is also wise to remember that minor and major operations are at times the starting point for effort syndrome. Vaccines and sera, intestinal and other antiseptics come and go, but the chronic cases are as resistant to improvement as ever.

Præcordial Pain.

The only other problem that time permits me to discuss is the interpretation and significance of præcordial pain. This embraces so many important aspects that no attempt will be made here to cover the whole ground.

Comparable to syncope, præcordial pain may be harmless or serious, but in either case, like syncope, it usually causes much perturbation to the patient. Pain in the anterior part of the left side of the chest is seized on by nervous subjects as being due to heart, while the sturdier types are prone to blame indigestion and both are commonly wrong. The former worry themselves needlessly and the latter court disaster through neglect or bravado.

Varieties of præcordial pain, harmless to life, include some non-cardiac causes, such as fibrositis. This is usually evidenced by tender points; it is made worse by arm movements and tends to improve with exercise, "warming up to it," but recurs after rest.

Referred pains from localized spondylitis should also be excluded. Pleural and thoracic conditions should be eliminated, especially tumours, aneurysm *et cetera*.

Being satisfied that the pain is of anginal nature, it is now necessary to determine whether it is the grave form or the harmless.

There are many classifications distinguishing between these two types such as the familiar terms *"angina minor"* and *"angina major"* "vasomotor angina," "pseudo-angina." The terms themselves are a source of confusion. Some use *"angina minor"* to mean the harmless variety, others as a less severe form of grave angina.

Personally, I think Mackenzie's scheme of two groups, "primary angina" and "secondary angina," is the most practical and helpful. Primary angina includes the serious forms based on arterial and cardiac disease. Secondary angina includes the harmless forms based on a hypersensitive nervous system. This in turn depends on neurasthenic and neurotic states produced by a similar variety of causes, which we saw to be the basis of a big group of effort-syndrome cases. Inherent or acquired nervous instability, neuroticism, the effects of suggestion, chronic anxiety and fatigue conditions derived from a most diverse selection of causes to which also add chronic toxæmia leading to neurasthenia and sometimes physical strain. There ensues a combination of psychological and physical disharmony which is a potent cause of ill-health; though often enough these individuals are regarded not so much as sick people as nuisances. And so they are to their relatives and friends and maybe to their physicians; it is usually "physicians," the plural. Nevertheless they require skilled management and much understanding. They are usually young men or women; the latter predominate. It is quite rare to find serious primary angina in young women, that is, excluding patients with obvious heart lesions. They have many vague symptoms indifferently localized, the pain itself being of an aching character or stabbing. It is not usually substernal and does not radiate. It comes on without sufficient exciting cause, often while the patient is at rest. The patients tend to be restless, rather than immobile as in grave angina. Attacks in bed may occur also in primary angina, but such cases are usually not difficult to diagnose. There is a certain looseness and inconsistency and obvious exaggeration in the description of the pain, which stamps the neurotic. A careful history, eliciting all incidents of the attack, may be a more important guide to correct diagnosis of the type of angina than the physical examination. As a matter of fact it does not often happen that the medical attendant sees the patient while in the attack and accordingly has to rely on the patient's description more than on physical signs of cardio-vascular disease.

In primary angina it is most important to get all the details of the first attack; as a rule the incident is related sufficiently graphically to leave little doubt as to the diagnosis. Initially the pain is usually substernal or præcordial and is severe enough to pull the patient up. There is a sense of substernal oppression, which may become a gripping constriction and may extend round the ribs. The

degree of apprehension is in direct proportion to the amount of pain and to the temperament of the patient. The pain may radiate down the left arm to any extent, sometimes no further than the shoulder or to the elbow or right down to the fingers. It may at times radiate across the chest and into the right arm; sometimes up into the neck and over the jaws and face; sometimes over the epigastrium.

Walking to work after a meal frequently brings on the pain. Flatulent distension and belching very often are accompaniments and getting rid of the gases from the stomach gives real relief. Hence air-swallowing is a habit soon acquired in an attempt to repeat previous successful relief of pain by eructations. "Wind around the heart" is the usual expression and a fervent wish and strong effort is made to get rid of it. This association of meals, flatulence and pain often leads to an erroneous diagnosis of indigestion by the patient and, let us confess, by the physician. The pain follows a well recognized distribution along the cervico-dorsal segments and is a good example of referred pain causing viscero-sensory and motor effects. Unusual distributions and radiations may be due to overflow of impulses to other segments and nervous paths.

Cunliffe Shaw[1] gives a nervous hypotheses for *angina pectoris,* based on experimental work on rabbits. It is claimed that chronic irritation to the cardio-aortic nerves produces various degrees of aortitis. Hence he holds "that *angina pectoris* is a group of symptoms primarily invoked by a nervous lesion, located either in the upper dorsal or lower cervical cords, its associated ganglia or their connexions, and that such lesion, secondarily invokes inflammatory and degenerative reactions in the aorta and possibly the heart," a vicious circle arises "which produces hypersensitivity of the sensorimotor mechanism, especially the vagal element, commonly resulting in sudden death in severe cases."

He regards surgical interference as justifiable, consisting in complete extirpation of the left cardio-aortic sympathetic nerve and connexions. It would be interesting for the pathologist to extend his examinations to these nervous structures. One of the most prominent advocates for surgical relief is the Roumanian, Daniélopolu.[2] Briefly his theory is that fatigue products in the heart irritate the sensory nerves and cause not only pain but reflex pressor stimulation. He contends that the proper treatment is to suppress the pressor circle by intercepting as many of the cardio-aortic sensory fibres as possible, whilst preserving the efferent fibres to the heart from the medulla and upper part of the cord, so as not to destroy the pressor mechanism altogether. Daniélopolu's book gives full anatomical details so far as they are known. Thirteen patients have been operated on; the results appear to justify the procedure. Jonnesco and others have also operated on nervous connexions, but their results are not encouraging. It sounds an heroic procedure and further reports are awaited with interest. Obviously a careful selection of patients would be very necessary. Another line of attack is to inject the dorsal ganglia. Speaking of primary angina, it needs to be emphasized that sudden death occurs only in about one-third of the patients. The dramatic and often tragic termination over-impresses us with its alleged frequency. Gradual heart failure with dropsy, progressive exhaustion, some intercurrent infection or an apoplexy *et cetera* accounts for the remainder.

There is no constant pathological change found in anginal patients. Arteriosclerosis is frequent, but by no means invariable. Similarly high blood pressure is often absent; indeed the pressure may be strikingly low, for example, a systolic pressure of 100 to 120 millimetres of mercury.

Most clinicians have been struck with the association of gall bladder disease, cardio-vascular degenerations and angina; though this inference needs to be qualified by the remainder that all these are commoner after middle age. However, it seems to be more than a coincidence. Chronic cholecystitis may act as a toxic focus damaging liver, pancreas and heart and it may be that the cardiac symptoms are more insistent than the quieter signs of chronic hepatitis and pancreatitis. Glycosuria is another frequent association, possibly from the pancreatitis. Stomach, duodenum and gall bladder also play their part reflexly, especially in producing extra-systoles.

Coronary Thrombosis.

In recent years attention has been focussed on violently severe prolonged attacks of *angina pectoris,* due to coronary thrombosis and cardiac infarction. The syndrome of symptoms is fairly characteristic and a diagnosis in life can be made with reasonable exactitude.

Recovery is possible, depending mainly on the size of the coronary branch involved and the degree of anastomosis, for the coronaries are not end vessels, as was formerly taught. The usual history of the case is that the patient, well past middle age, has been subject to pain in the chest on exertion, especially after meals, for some time past. Consequently a diagnosis of indigestion is frequently made by a patient. After a longer or shorter history of such attacks a violent agonizing pain comes on, most commonly whilst the patient is in bed. The pain is excruciatingly severe, far exceeding anything in the patient's previous history. There is definite apprehension of impending dissolution; in fact, so unendurable is the agony that the wretched victim gasps out for relief, even by death.

The pain is substernal or over the præcordium and may or may not radiate into the left or both arms. Sometimes it is epigastric and as there may be signs of collapse and vomiting it closely resembles an acute abdominal crisis. The appearance of the patient is one of shock and urgent distress. He prefers to be propped up with pillows, but is fearful of the slightest movement and any attempt at such

causes considerable dyspnœa. The face is grey and the expression is one of acute anxiety, often made vocal in short gasping sentences about the pain and requests for relief. Frequently there is a cold sweat and attempts at expulsion of flatulence; sometimes there is retching and vomiting.

The pulse is weak in volume and often in definite contrast to the full high tension pulse which may have been observed on former examinations. It is frequently irregular from extra-systoles and often slow, about sixty per minute.

The blood pressure shows a striking fall in systolic reading, even down to sixty to eighty millimetres of mercury. The diastolic pressure is difficult to register; the pulse pressure is obviously small. The heart sounds are feebly heard. If an anterior coronary branch is involved, the inflammatory reaction causes a small patch of pericarditis and local friction rub may be heard. There may be a mild rise of temperature to about 37·7° C. (100° F.) and a slight leucocytosis which may lend colour to the picture of an upper abdominal emergency. The pain is not relieved by simple means, such as brandy, hot drinks, mustard plasters *et cetera*, nor by the usual vaso-dilator remedies as amyl nitrite, trinitrin *et cetera*. An injection of fifteen milligrammes (a quarter grain) of morphine may even fail to control the attack and the dose frequently has to be repeated; however, it is the best drug on which to rely. Absolute rest is very important and too much medication should be avoided. Digitalis and strychnine are unlikely to do good. Diuretin or calcium diuretin are supposed to be coronary dilators and may be tried in doses of about 0·45 gramme (seven and a half grains) three times a day. The prognosis depends on the size of the coronary branch involved and whether the heart can keep going sufficiently long to survive the crisis. If the acute symptoms are surmounted, it is obvious that prolonged rest is necessary to allow the heart muscle to recover more or less. Permanent damage may result, but good recovery has been recorded in cases which seem typical. They usually show electrocardiographic evidence of a damaged myocardium. Of course the presence of cardiac infarction in cases of recovery is by inference only.

The syndrome has been worked out by correlating *post mortem* evidence of coronary thrombosis with case histories and is an interesting example of clinical pathological research. The condition is not very frequent. In the last twelve months I have seen in consultation only eight patients in whom I have ventured to diagnose angina from coronary thrombosis. Of these seven died, the other one is comparatively well. No *post mortem* examinations were obtained in this small group.

One of the most interesting cases was that of an ex-sea captain, aged fifty years, whom I saw with Dr. A. H. Crowley. During war service he suffered from a form of *endarteritis obliterans* of the legs which may have been Buerger's disease or *thrombo-angiitis obliterans*. Eventually the right leg had to be amputated in 1922. He was able to do office duties, though suffering from intermittent limp in the other leg. His blood gave no reaction to the Wassermann test. In June, 1927, he had a very severe attack of angina, coming on at 2 a.m. and lasting some time in spite of morphine. His pulse progressively slowed from 72 per minute to 50 and then to 36 per minute; the temperature was 37·7° C. (100° F.) and respirations 36, the blood pressure 100/60 millimetres of mercury and the heart sounds were scarcely audible. The progressive pulse slowing argued an involvement of the bundle of His causing heart block. This structure is supplied by a branch of the right coronary artery and it is reasonable to suppose that this was the localization of the thrombotic process. The heart rate gradually returned to 72 per minute. He survived this attack and a month later was able to have electrocardiograms taken which showed evidence of myocardial damage.

The blood pressure remained at 100/70 millimetres of mercury. Subsequently he was admitted to the Repatriation Hospital, where he had two more attacks, with the same phenomena of slow pulse and died in an attack. Permission for *post mortem* examination was not obtained.

Another case was that of a male, aged sixty-four, who had recurring angina for four years. He had an enlarged heart and signs of aortic and mitral valve disease. His systolic blood pressure averaged 160 and his diastolic 100 millimetres of mercury. After a very violent nocturnal anginal attack lasting many hours and needing two injections of morphine, the blood pressure fell to 120 millimetres systolic, 100 millimetres diastolic and remained low till the end, three months later. After the fall of pressure he had less severe angina, but developed progressive dyspnœa and dropsy and obvious myocardial exhaustion. He died suddenly in the night.

Of the eight patients, only one was a female, aged sixty, whom I saw with Dr. F. May. She showed the typical syndrome and Dr. May informs me that she developed a left-sided hemiplegia four days later, followed by evidence of pulmonary thrombosis. She died within one month.

The average age of the eight patients was fifty-eight, ranging from forty-seven to seventy. Space does not allow of full histories which were strikingly similar and quite typical of the descriptions already given.

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PYELITIS OF INFANCY: A PLEA FOR FURTHER INVESTIGATION.

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THERE can hardly be any disease about which the ideas of the majority of the profession are more firmly fixed than is the case with pyelitis of infancy. The majority of practitioners seems to be agreed that the symptoms of this disease are caused by the invasion of the mucosa of the kidney pelvis by pus-forming organisms, generally the *Bacillus coli communis*, and that the disease is at least twice as common in girls as in boys. The object of this paper is not to give a full account of pyelitis, but to attempt to show that what seems to be the general belief regarding the pathology of this disease, is not

fully explained by the facts and that further pathological investigation is necessary before it can be said that the condition is fully understood. The following remarks are intended to apply only to cases of pyelitis in infants under the age of two years.

Mode of Infection.

A great deal of discussion has taken place as to whether the responsible organisms gain access to the pelvis of the kidney by direct spread up the urinary passages or by the general circulation. Very many at least hold to the view of direct spread by the urinary passages and the greater liability of the vulva and urethra of girls to contamination by fæcal matter is brought forward to explain the greater frequency of pyelitis in girls than in boys. In other words many believe that what is known as an ascending infection takes place. Now it seems to me that direct spread of infection by the urinary passages can take place against the direction of the urinary stream only if reversed peristalsis be present. Reversed peristalsis in the ureter in the absence of obstruction has, as far as I know, never been demonstrated and in the majority of infants suffering from pyelitis there is no obstruction to the flow of urine.

Again, the bladder mucosa is very prone to infection by the *Bacillus coli communis*, the causal organism in most cases, and therefore it is fair to assume that if organisms reach the pelvis of the kidney by direct spread by the lumen of the passages, cystitis would be very frequently found. This is not the case. The urine in pyelitis is most often acid in reaction and not alkaline, as it would be in cystitis. Also it is frequently the case that cystitis clears up without the development of pyelitis.

With regard to the possibilities of an ascending infection by way of the blood vessels of the ureter, the abdominal portion receives its blood supply from the renal and the spermatic arteries; the course of the circulation here, therefore, is from above downwards and not from below upwards. Ascending infection by way of the lymphatics of the ureter is a possibility, but has never been proved and more work must be done on this point before dogmatic statements can be made.

The belief that pyelitis is very much commoner in girls than in boys is, as I have already remarked, brought forward as an argument in favour of ascending infection. The very universality of this belief may have the effect of perpetuating it. A practitioner holding this belief would be more likely to look for the condition in girls and there would be the possibility of an accurate diagnosis being made more often in the case of girls than in boys.

If figures were available, the death rate from pyelitis would be likely to give us an accurate idea of the incidence of the disease in the two sexes. I regret that so far I have not been able to find any accurate statistics on this point. The only figures I have so far found are contained in a note of one hundred and five cases of pyelitis in infants from the Harriet Lane Home. The death rate in this series is given as 60% girls, 40% boys. These figures would seem to indicate that the disease is commoner in boys, that is the general belief and surely an ascending infection in boys must be very rare.

Infection by Way of the General Circulation.

The peculiar nature of the circulation through the kidney makes it more liable than any other organ in the body to become the seat of focal infection through the general circulation. All the arteries supplying the parenchyma are end arteries. No anastomosis whatever takes place between one blood vessel and another. Furthermore it has been found impossible to reverse the circulation through the kidney, so that occlusion of a blood vessel in the parenchyma absolutely shuts off a portion of the kidney substance from the circulation and provides an opportunity more favourable than can be obtained anywhere else for the development of microorganisms. The renal artery, however, supplies the pelvis of the kidney as well as the parenchyma, but here the circulation is of a more usual nature; the arteries are not end arteries and anastomosis takes place between the various branches.

If infection does reach the kidney through the renal artery, why should the pelvis be infected so frequently and the kidney substance escape? All the anatomical evidence seems to show that the reverse should be the case and that the pelvis should be very much more resistant to infection by this route than the kidney itself.

It would be as well here to analyse the available literature on this question to see whether the anatomical possibilities are confirmed by investigations which have already been carried out.

When one examines the literature dealing exclusively with infants under the age of two years, two facts are apparent: first, that comparatively few papers have been written dealing exclusively with infants and second, that in very few indeed of the cases described has a complete histological and bacteriological examination been made. In some the authors have apparently relied on naked eye examinations of the urinary tract for the conclusions they have drawn. Now it is a notorious fact that naked eye examinations of the pelvis, ureters and bladder are of very little value on account of the very rapid desquamation of epithelial cells which so often takes place after death. Therefore, I think that in forming any conclusion from the literature importance should be given only to those cases which have been completely examined histologically and bacteriologically.

It is manifestly impossible in a paper such as this to go in detail into all the papers contained in the bibliography of the subject, but a careful perusal reveals the rather astounding fact that as far as infants under two years of age are concerned, the literature seems to contain only nine cases which

have been examined in detail. Of these two were reported by McDonald in 1909 and seven by Thiemich in 1910. McDonald's two patients were both boys, four and seven months old. Thiemich reported three boys and four girls, the eldest child in his series was eight months old. The finding in eight of the nine cases was suppurative nephritis of a focal nature and in the remaining case cystitis.

Both Frew and Hutchison in London had noted three years ago focal suppurative lesions in the kidney in some of their cases and I see from the notes which I took of their lectures, that they were both of the opinion that the disease began as a pyelitis and in cases which cleared up, the kidney substance was not infected but that in fatal cases the kidney substance became infected secondarily to a pyelitis. If this were true, one would expect to find pyelitis present in those nine cases of which we have a detailed account, as there is no reason that I know of why pyelitis should clear up when the infection has spread to the kidney. Pyelitis, however, was not found or recorded as a cause of death in these nine instances.

Focal Infection.

There is a point which must have been noted by many who work among children and that is that in many cases of infants dying from such a disease as gastro-enteritis, pus will be found in the urine of very many of them a day or so before death. This happens with too great frequency to be explained by accidental contamination of the urinary passages by fæces. I am well aware that the view is held by many observers that *Bacillus coli communis* can traverse the wall of the bowel in certain diseased conditions and directly infect the pelvis of the kidney. This may be so, but one fact makes me very much doubt whether this is the explanation of the appearance of pus in the urine in these cases before death and that is the fact that if the middle ear be opened at *post mortem* examination, muco-pus will be found there also with surprising regularity.

The view that appeals to me most is that in cases which are going on to a fatal termination, a stage is reached when the patient is overwhelmed by his infection. Organisms freely enter the circulation and the pyuria and the middle ear suppuration may be looked on as terminal events secondary to the initial lesion.

This view raises the interesting question as to how many of our cases of pyuria in infancy are due to a focus of infection elsewhere? It seems likely that if more cases could be fully investigated many patients would be found to have a focus of suppuration from which the kidney condition had arisen.

There is just one further point which I wish to bring forward in favour of this disease being rather a suppurative focal nephritis and that is the character of the temperature seen in cases of so-called pyelitis. The chart in these cases shows a definitely swinging temperature and we commonly find a symptom, very rare in infants apart from malaria rigor. In the rare cases of calculus in infants associated with pyelitis it has not been my experience that the temperature chart was typical, nor have I seen rigors as a prominent symptom. On the other hand, I can recall several cases of trauma of the kidney substance associated with definitely swinging temperature and rigors, also one case in which a calculus was present. In this instance the character of the temperature changed completely after the removal of the stone, when some damage and infection of the kidney substance had occurred at the operation. My experience has led me to believe that damage to the kidney substance, whether traumatic or infective, is often associated with a temperature such as we have come to expect in cases of the so-called pyelitis of infancy.

Conclusion.

In conclusion, it is extremely difficult to see on what foundation our belief is rested that pus in the urine of an infant means pyelitis or how that belief as the name of the disease implies, ever became so wide as it is today. The evidence available seems to point more to a focal suppurative nephritis as the cause of symptoms.

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DIATHERMY APPLIED TO MEDICINE.[1]

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I REGARD it as a compliment that you have asked me to give you my experiences as to the results of treatment by diathermy and hope to advance your interest into lines which I think to many will be entirely new.

Treatment by diathermy has for some time past been more or less confined to surgical work in which it has been employed for its destructive properties. Its next stage of usefulness was to take the place of radiant heat. I found it of great benefit in such conditions as lumbago, sciatica and painful joints affected with arthritis or for the absorption of fluid in joints. Its action in dispersing the swelling due to recent injury with extravasation and the consequent relief of pain is truly remarkable. It is not my purpose here to go into a history of diathermy or dilate upon what can be found in the ordinary textbooks, but rather to give you the results of treatment in certain directions which I believe to be new or known to only a few.

In the use of diathermy the heat factor is the one which I believe to be responsible for the results obtained; the action of the current I consider to

[1] Read before the Section of Electrotherapeutics of the Victorian Branch of the British Medical Association on May 17, 1928.

be negligible. Heat is obtained by a current alternating between the two poles at a rate of somewhere about a million times a second, such alternations being imperceptible to the tissues. This heat being generated within the tissues, is different from that obtained from radiation, as the latter does not penetrate to the deep tissues. Its main action is to cause a definite dilatation of the blood vessels resulting in an enormously increased circulation throughout the part under treatment. When employed through the body or along the limbs, a decided rise of temperature is experienced. Exactly what physiological changes take place in the part when under diathermy I cannot say, but experience has shown that absorption of inflammatory products occurs. This is not confined to recent inflammatory products, but affects to a certain extent past ones also. With the advent of an increased blood supply increased facilities for the repair of tissues are provided and greatly advanced powers of defence against infecting agents.

My first experience with diathermy was a personal one when I received great comfort and improvement in an abdominal condition with many adhesions. From this I was led to believe that adhesions actually become absorbed. After this I had a very good demonstration which must confute the ideas of all sceptics on this matter. A patient of mine who some years previously had undergone a serious abdominal operation, had the abdominal scar anchored down to her lumbar spine. The adhesions caused a dipping in of the scar with fixture. This resulted in great difficulty in defæcation and there were frequent occasions when she had to be rushed into hospital and receive very drastic treatment to free the bowels. She was advised to have a course of diathermy and after a certain number of treatments had been administered, the adhesion gave way and the abdominal wall became free and lax. From that day there has been no further trouble with the bowels. This adhesion appeared on the film as a distinct band. Needless to say I was extremely impressed by this result, as it opened up the question as to the extent to which diathermy could absorb adhesions and fibrous tissue laid down in organs as the result of chronic inflammation. I therefore determined to apply diathermy in various conditions and to ascertain what results could be obtained. In July, 1925, a man, aged sixty-nine years, walked into my rooms in much distress; his face was ashen with drops of perspiration on his forehead; he was short of breath and had great cardiac pain. He told me he had had heart trouble for four years. Examination revealed a very enlarged heart with auricular fibrillation; his pulse rate was 162, his systolic blood pressure was 152 millimetres of mercury and his diastolic pressure 100. He had severe pyorrhœa and sinusitis and a history of rheumatic fever at the age of twenty-eight. The myocardial condition was more pronounced than the valvular one. The first idea that occurred to me was that if the coronary circulation of this heart could be improved, he would get some definite relief. I therefore applied diathermy tentatively and waited to see what would happen. In a very short while he said that the pain had eased considerably and it was not long before it had ceased altogether. He was put on the usual digitalis and such treatment as is employed in these cases. In about ten days his pulse rate was eighty. This man had been under treatment off and on during the previous four years with digitalis. Perhaps the most striking thing was the rapidity with which his skin took on a nice pink tone. This has been repeatedly noticed in other cases. The diathermy treatment was continued twice a week for two months and it was not long before he was back in his office where he had not been for a long time; he was able to spend his week-ends in the hills attending to a little place he had there. From then up to the present he has maintained his good heart condition. There is no doubt that the clearing up of his pyorrhœa and nasal trouble helped him a good deal, but both he and I were perfectly satisfied as to the benefit he received from diathermy. What more rational idea could one have of improving the coronary circulation and possibly removing some of the fibrous tissue laid down amongst the muscle fibres?

During the last three years I have had ample opportunity of employing diathermy in similar cases and there are now men going about with fibrillating auricles and rocky hearts who without diathermy would have been dead long ago.

When examining chests with the fluorescent screen I have been struck by the enormous amount of fibrous tissue that is laid down in the lungs, particularly in cases of chronic bronchitis. The process of the laying down of this fibrous tissue is not universally known. It is almost invariably the result of nasal infection. Chronic nasal infection is very common, in fact 25% of people attending medical clinics for other diseases have it. The drainage from these infected sinuses is along a chain of glands on the posterior border of the sterno-mastoid down the neck to the mediastinal glands. From there infection spreads along the lymphatics round the bronchi and often leads to chronic bronchitis and is occasionally accompanied with asthma. Experience already gained led me to try the effect of diathermy in these cases and without exception the results were uniformly good. Cases of bronchitis and asthma which had been going on for years, cleared up in a remarkable way and the patients have remained well. A child with a lung abscess or localized empyema communicating with a bronchus, prior to treatment with diathermy every few weeks became feverish and expectorated a collection of pus. Her condition cleared up and there has been no recurrence of her trouble. Persons with chronic cholecystitis and even those with gall stones have claimed great improvement and freedom from pain and discomfort greater than that received as a result of any other form of treatment. The same may be said of chronic appendicitis. In patients with diabetes it is invariably employed by me, as I feel sure that it increases the nutrition of the tissues.

There is no treatment that gives such relief to chronic pelvic troubles as diathermy, be they dysmenorrhœa, adhesions or chronic tubal conditions. In one case of multiple gonorrhœal arthritis of long standing great benefit was obtained, the electrodes being applied so as to heat up the whole pelvis as there was every indication of chronically infected tubes. Another patient with exophthalmic goître and an infected cervix recovered after receiving cervical treatment. I think that as much heat can be applied through the pelvis as with one pole in the cervix. In cystitis with great pain on micturition the relief is very rapid and all acute symptoms soon clear up.

One of the most difficult of cases we are called upon to attend to is the chronically infected prostate. As a focus from which other disease is produced, this is commoner than is usually recognized. Whether the treatment be for infection or enlargement alone, if it be possible to get an enlarged prostate without infection, various methods have been employed, such as massage, X rays, high frequency current and diathermy. Having tried all and various combinations of them, none has given me such good results as diathermy with an occasional treatment by X rays. One very seldom fails to give relief to the sufferer with enlarged prostate who has to be in and out of bed half a dozen times in the night. A few years ago a captain in the pilot service came to me suffering from exophthalmic goître, having been declared unfit for further service. After obvious infections in his nose and throat had been removed he still failed to get right. He had always disclaimed any prostatic trouble, but on massage a copious amount of infected material was brought away; with the aid of a vaccine and diathermy he rapidly got well. After some persistence he managed to get reinstated and has been at his old work ever since.

With hæmorrhoids in the acute stages with much pain diathermy gives great relief and a judicious combination of injection and diathermy treatment is sufficient to cure nearly every patient who presents himself.

Last November a man, aged forty-one years, consulted me for pain in his legs chiefly in the calf muscles, which came on soon after he started to walk. This had progressed to such an extent as to prevent him carrying on his work. In a week with daily treatments he was able to walk about town with a fair degree of comfort and at the end of a fortnight he went home having lost all discomfort. He returned a week or two later and had two or three further treatments, as he had a suggestion of the old pain. Since then I have heard nothing from him, but I feel sure that he would be back quickly if his trouble recurred. What more rational treatment could there be for intermittent claudication than diathermy?

On March 14, 1928, a man, aged fifty-one years, consulted me for high blood pressure. He had been under treatment by his local doctor, his systolic pressure having been 260 millimetres of mercury for some considerable time. He looked a very sick man, pale faced, short of breath on exertion and with his apex beat outside the nipple line. His urine contained a definite cloud of albumin, but was somewhat concentrated. He had some pyorrhœa and tonsillar infection. Starting cautiously I gave him diathermy through his kidneys and he experienced relief very rapidly, the first result being a greatly increased amount of urine. Within a fortnight the systolic pressure had gradually dropped to 210. Beyond six centigrammes of thyreoid extract three times a day and some digitalis he received no drugs to reduce his pressure. The albumin was quickly reduced to a trace and has now gone. His colour is pink, he can walk about without any discomfort and the apex beat has returned to normal position. He has had some teeth extracted and treatment given for his infected tonsils, which has no doubt helped his kidney condition. His outlook on life from being that of a man faced with the inevitable has completely changed and he is now looking forward to a renewed state of health and resumption of work. When we picture to ourselves the pathological condition of his kidneys, is there any saner form of treatment to suggest than to dilate the vessels and help to reduce the fibrous tissue deposited there?

In the Year Book on the section of general therapeutics by Fantus, which has only this day come to hand, I see an account of some experimental work done by Weinstein and Klein on the secretion of urea and chlorides after diathermy had been applied to the kidneys. The results showed a definite increase of both chlorides and urea in normal patients, while in one with chronic nephritis and considerable hypertension the urea was more than doubled after the treatment.

These cases are selected as types of the conditions we physicians are called upon to treat; when we consider that the chief cause of damage to body tissues is brought about by bacterial infection and the resulting response on the part of the body leaving behind it fibrous tissue, irrespective of what organ is involved, judicious application of diathermy will help to restore those tissues to their former condition. To those who have had no insight into the action of diathermy, this will sound a gross exaggeration of facts, but the matter can be proved by experiment by those who are progressive enough to try.

It behoves us as physicians to equip ourselves with all the means which will be useful to us in the treatment of disease and we have at our command today forces which were undreamed of a few years ago. Of these diathermy stands out as one of the most useful. It will reduce the necessity for operation in many cases, particularly in connexion with gall bladder conditions, in which I have been much impressed by the unfailing benefit derived.

Dr. Frecker, of Kiama, New South Wales, has recently published a method of dealing with tonsils by means of diathermy. Since the publication of

his article I have given it a good trial. The mass of the tonsil can unquestionably be destroyed by this method and should there be any lurking infection left, this can easily be dealt with by ultra-violet rays and the high frequency currents. I have for long been employing X rays to help in the resolution of the tonsillar tissue. This is no doubt efficacious, but it takes time. Diathermy is much more rapid, as half a large tonsil can be destroyed at a sitting. I fully believe that the combination of diathermy with ultra-violet rays and some high frequency current will solve the problem of dealing with tonsils in adults. Where tonsils are of moderate size they can be dealt with in one sitting. Under these conditions the reaction is almost as much as if the tonsils had been enucleated, although I have never seen the soreness last more than three days. This I believe to be the best procedure, as each tonsil can be dealt with completely at one sitting.

In conclusion I may say that in diathermy we have one of the most powerful forces at our command in the treatment of disease and a great help in restoring damaged tissues to something approaching their former condition.

THE ESTIMATION OF UREA IN BLOOD.

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AND

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Biochemist of the Institute of Pathological Research, Royal North Shore Hospital of Sydney.

SEVERAL methods are available for the estimation of blood urea, most of them depending on the fact that the enzyme urease, present in jack bean and soya bean, quantitatively converts urea into ammonium carbonate. The urease methods fall naturally into two classes: those in which whole bean meal is used and those in which the enzyme is used after preliminary separation from the inactive portion of the meal. In the first class is Maclean's method[1] and in the second are the methods generally adopted in America, due chiefly to Myers,[2] Folin and Wu[3] and Van Slyke and Cullen.[4] In the former class whole blood is used, but in the latter either whole blood or a protein-free filtrate of blood is employed. In all cases ortho-, pyro- or meta-phosphates in the form of their acid salts are added both to insure that the ammonium carbonate produced by the urease will be neutralized and the solution remain slightly acid in reaction and also to provide a phosphate solution in which urease appears to exert its maximum enzymic activity.

A specimen of de-fatted "Arlco" jack bean meal was found during a long period to give by Maclean's method results for the urea content of blood judged as correct by a comparison with the non-protein nitrogen content. In some cases normal urea figures were confirmed by the results of the urea concentration test. A new lot of the meal of the same brand, however, was found after a time to give results obviously too high, as did a third lot from the same source. To investigate this point more fully specimens of normal and nephritic blood were analysed for urea by Maclean's method with the use of jack bean meal and also in some cases freshly ground soya bean. Analyses for urea were also done by Myers's method and the non-protein nitrogen was estimated also. In normal blood the ratio of urea nitrogen to non-protein nitrogen is approximately 50%, but in nephritis the percentage may be greater.

The results obtained are given in Tables I and II, in which it is to be noted figures are given for urea nitrogen and not for urea. The ratio of urea nitrogen, as estimated by Myers's method, to the non-protein nitrogen is seen in Table I to be normal. When, however, results given by Maclean's method when jack bean meal is used, are considered, it is at once seen that they are erroneous for in all cases the ratio of urea nitrogen to non-protein nitrogen is much greater than the normal ratio. In some cases it is seen that the urea nitrogen is even greater than the non-protein nitrogen, an obviously impossible result. The jack bean meal responsible for these incorrect values had been giving satisfactory results for some time before it commenced to deteriorate. Blank determinations by Maclean's method with 0·40 gramme of one specimen of the jack bean meal which gave incorrect results, yielded seventeen milligrammes of nitrogen. Evidently a change may occur in jack bean meal on ageing that renders desirable a check on the urea content of blood, as estimated by Maclean's method, by the simultaneous estimation of non-protein nitrogen.

In our opinion it is preferable to use for the estimation of blood urea an alcoholic solution of urease as required by Myers's method. The urease solutions which were used in the work, were found to retain their enzymic activity for at least six weeks when kept in the ice chest. That the deterioration of the jack bean meal does not interfere with the preparation of a satisfactory urease solution from it has been shown by the fact that the results given were obtained by the use of a urease prepared from a deteriorated meal.

It has been pointed out by Hindmarsh[5] and Sumner and Hand[6] that the urease content of different varieties of soya bean and different samples of jack bean varies greatly. In investigating the urease activity of eighteen varieties of soya bean, Hindmarsh found variations up to 50%. By the courtesy of Miss E. M. Hindmarsh, of the University of Sydney, we obtained a variety of soya bean known as *Ito San* which she had found consistently satisfactory. Our results (in Table II) with this bean freshly ground, show that the figures it gives with Maclean's method conform to the figures given by Myers's method and to the non-protein nitrogen

TABLE I.

Case.	Milligrammes per 100 Cubic Centimetres of			Urea Nitrogen (Myers's) / Non-Protein Nitrogen Percentage.
	Urea Nitrogen [1] (Maclean's Method) (Jack Bean).	Urea Nitrogen (Myers's Method).	Non-Protein Nitrogen.	
1. Nephritis	50·5	32	56	58
2. Nephritis	44	27	43	63
3. Nephritis	32	21	35	60
4. Normal	34	17·5	34·5	51
5. Normal	24	10·7	20·5	52
6. Normal	29·5	14	29	48
7. Normal	28·5	14·5	32	45
8. Normal	36·5	14	31	45

[1] Urea-nitrogen figures may be converted to urea by multiplication by the factor 2·14.

TABLE II.

Case.	Milligrammes per 100 Cubic Centimetres of			Urea Nitrogen (Myers's) / Non-Protein Nitrogen Percentage.
	Urea Nitrogen (Maclean's Method) (Soya Bean, Ito San).	Urea Nitrogen (Myers's Method).	Non-Protein Nitrogen.	
1. Normal	18·2	17·5	34·5	51
2. Normal	11·1	10·7	20·5	52
3. Normal	14·7	14	29·1	48
4. Normal	15·2	14·5	32	45
5. Normal	27·9	26·6	—	—

results. However, difficulty is frequently met in obtaining a satisfactory soya bean for the purpose of estimating blood urea and in our experience a urease solution as prepared below gives consistently accurate results.

Experimental Details.

The following method of preparation of urease solution was adopted:

Three grammes of "Permutit" powder in a flask were washed by decantation, once with 2% acetic acid and twice with water. To the moist "Permutit" were added 100 cubic centimetres of 15% alcohol and five grammes of jack bean meal. After continuous shaking for fifteen minutes the mixture was filtered and the filtrate, containing the urease, was stored in the ice chest.

The acid phosphate solution for use with the urease consisted of 140 grammes of sodium pyrophosphate and twenty grammes of glacial phosphoric acid dissolved in water and made up to one litre.

In the estimation of urea by Myers's method[2] one cubic centimetre of urease solution, two drops of the phosphate solution and two cubic centimetres of blood were incubated at 50° C. for fifteen minutes in a tube fitted for subsequent aeration of the mixture. At the end of this period five drops of caprylic alcohol and five cubic centimetres of saturated sodium carbonate solution were added and the liberated ammonia was aerated into twenty-five cubic centimetres of $^{n}/_{100}$ sulphuric acid. When aeration was complete, the ammonia was determined either by titration of the sulphuric acid with $^{n}/_{100}$ sodium hydroxide in the usual way or by nesslerization and colorimetric comparison with a standard, both of these variations being found to give the same result.

The micro-Kjeldahl digestion for the estimation of non-protein nitrogen was done according to the technique of Myers.[7]

Summary.

1. Certain specimens of prepared jack bean meal have been found to give too high results when used in the estimation of blood urea by Maclean's method.

2. An alcoholic solution of urease prepared from these specimens of jack bean meal gives correct results for the urea content of blood estimated by Myers's method.

3. A variety of freshly-ground soya bean used according to Maclean's method also gives correct results for the urea content of blood.

References.

(1) H. Maclean: "Modern Methods in the Diagnosis and Treatment of Renal Disease," Second Edition, page 51.

(2) V. C. Myers: "Practical Chemical Analysis of Blood," Second Edition, page 45.

(3) O. Folin and H. Wu: "A System of Blood Analysis," *The Journal of Biological Chemistry*, 1919, Volume XXXVIII, page 81.

(4) D. D. Van Slyke and G. E. Cullen: "A Permanent Preparation of Urease and its Use in the Determination of Urea," *The Journal of Biological Chemistry*, 1914, Volume XIX, page 211.

(5) E. M. Hindmarsh: "Variations in the Urease Content of Different Varieties of Soya Bean," *The Australian Journal of Experimental Biology and Medical Science*, September 16, 1926, page 167.

(6) J. B. Sumner and D. B. Hand: "Crystalline Urease, II," *The Journal of Biological Chemistry*, January, 1928, page 149.

(7) V. C. Myers: "Practical Chemical Analysis of Blood," Second Edition, page 43.

Reports of Cases.

AN UNUSUAL BREECH PRESENTATION.

By J. GOLDMAN, M.B., Ch.M. (Sydney),
Lowood, Queensland.

A YOUNG woman, aged twenty-three years, was admitted to Lowood Private Hospital on March 31, 1928. On examination she was found to be in labour, the *os* was almost fully dilated and the membranes had ruptured. This was her second pregnancy.

Abdominal palpation and vaginal examination revealed a breech presentation, the presenting part being fairly high up and what was regarded as a foot being palpable.

After an hour and a half the presenting part had advanced well down into the pelvis and on vaginal examination I was very surprised to find a hand in front of and at the left side of the presenting buttocks.

Accordingly I diagnosed a frank breech presentation with a prolapsed hand and under ether anæsthesia I brought down a leg. The fœtus was born as far as the umbilicus shortly afterwards, the right arm being found wrapped around the back which was grooved as if a tight rope were tied around it.

The left arm was extended and had to be brought down. The pulsation in the cord now ceased and I had to hurry to extract the head. This was effected by the Smellie grip.

The infant was in a state of *asphyxia pallida*, but eventually responded to treatment and began to cry lustily.

Except for a swollen right hand (due to the *caput succedaneum*) I could find nothing abnormal about the child, a male 4·3 kilograms (nine and a half pounds) in weight. The mother had an uneventful puerperium.

The first confinement had been a breech birth and the child, a four kilogram (nine pound) female, was born alive. The patient was in labour thirty-six hours with her first and twelve hours with her second. The mother stated that she herself was born "feet first."

I would be pleased to hear of any similar cases.

Reviews.

MIND AND MATTER.

AUSTRALIANS will need no introduction to Professors Wood Jones and Stanley D. Porteous, who have in "Matrix of the Mind" enhanced their great reputations.[1] In the authors' words: "The book is the first fruit of correlated studies made possible by a grant from the Rockefeller Foundation to the University of Hawaii" and its aim is to blend the subject matter and viewpoints of the two sciences which have the same physical structure as a basis, namely, neurology and psychology. The very difficult goal is approached very happily and students will revel in the added interest which is given to both by the correlation.

The authors have the knack of asking pertinent questions, whilst the reader wonders why this aspect did not occur to him. As an example, instead of baldly referring to the formation of the neural tube, they demand why it is that a strip of the skin of the mid-line of the human back is turned in to form a central nervous system and they give as the reason that it is an area in which nothing else is happening. That the anterior cells should be motor and the posterior ones sensory is apparent since the cell last tucked in is the most recent recruit to the central nervous system and therefore in closer touch with the surface.

As the story unfolds, the authors frequently delve into the unexpected. "The outside of the body," they say, "is what others can see, is in fact what we look like. The internal nervous system dictates our actions and our conduct and is in fact what we are . . . . dual parts of one integral whole." That the bird which has a gay exterior has a behaviour entirely in accordance with his bright livery, is shown by a comparison between the robin and the sparrow. The human analogy is obvious.

Referring to reasons for the poor mental development of the *invertebrata* and their apparent incapacity to progress far along the evolutionary line, the authors point out that they made a fatal mistake when they started to build a brain around an œsophagus, because it could not grow without making feeding difficult and those more advanced had to be blood suckers. As for the marsupials, poor development of the *corpus collosum* was an insuperable bar to progress.

Discussing cerebral topography, the authors show that the manner of fissuration of the cortex follows the lines of differing areas of cortical structure. An interesting chapter on the relevant stimulus describes how the so-called blind worm is able to notice distinctly a putty coloured slug upon which it feeds, but does not appreciate other moving objects.

The rise and fall of the smell brain makes interesting reading, as does the portion of the book dealing with the growth of the visual centres. It is shown that the great assets of the human stock lie in the fact that no one element in the sequence was selected for undue specialization; the changes in hand and eye and brain have been coordinated into a harmonious growth. Human beings have not overdone the eye development, as *Tarsius* has; they have not unduly emphasized the hand, as has *Chiromys*.

As an example of the kind of problem dealt with, may be cited the explanation of the representation of areas in the motor cortex. The hand is near the face because the knowledge of the body passes in evolution from the muzzle to the hand. A reference to a standard work on anatomy shows that the *flexor carpi ulnaris* has as its function merely flexion of the wrist and slight adduction of the hand. Wood Jones and Porteous analyse this and find that the muscle may act as an agonist, an antagonist, a synergic or a fixator. The why and therefore of this complicated mechanism is a phase of the "new anatomy" which makes profitable reading.

The authors write engagingly on the various pathways to the brain and it is refreshing to read a description of such a puzzling tract as the lenticulo-rubro-spinal tract in the homely phraseology of a private path to the *corpus striatum*.

The book deals with such topics as facilitation, stimuli and responses, habits, monotony and interest, sense of aliveness, enjoyment and appreciation, the cortex and intelligence—a varied collection of phenomena about which space does not permit of present discussion. Recourse must be had to the book itself which is worthy not only of a prominent place on the library shelf, but of close inspection by all who are even remotely interested in that perennial wonder, the relations between mind and matter.

[1] "The Matrix of the Mind," by Frederic Wood Jones and Stanley D. Porteous; 1928. Honolulu: University Press Association. Royal 8vo., pp. 457, with illustrations.

A CLINICAL HANDBOOK.

THE stream of American publications flows unabated. "Physical Diagnosis,"[1] by Dr. W. D. Rose, is yet another on the simpler parts of clinical medicine. This author

[1] "Physical Diagnosis," by W. D. Rose, M.D.; Fifth Edition; 1927. St. Louis: The C. V. Mosby Company. Demy 8vo., pp. 819, with illustrations. Price: $10.00 net.

describes the clinical anatomy of the chest and abdomen; the elements of physical diagnosis by inspection, palpation and auscultation; the clinical pathology and morbid signs of "the commoner diseases of the respiratory and circulatory systems." Familiar subjects are handled well. We single out for special praise the section on the pulse, so often neglected in recent books, at least in comparison with the elaborate accounts given in older ones; our author describes not only the signs to be discovered by the finger, but also the use of the polygraph and the sphymograph. The differential diagnoses of ordinary diseases are copiously treated, but less common subjects, as tumour of the lung, very scantily; difficulties such as mediastinal tumour and mediastinitis are hardly noticed. The sections on the head and neck and on the nervous system are too short to be of much value and other subjects usually discussed in such works are omitted. We seek in vain for information on the ophthalmoscope, the laryngoscope, radiography, the bacteriological and chemical method of the laboratory. The English is remarkably good, surprisingly so in a foreign work, and we were not affronted by one misprint. The illustrations, mostly photographic, are numerous and beautifully produced, but often unnecessary, for instance that of "auscultation of the femoral artery." The printing, paper and binding are all good.

THE CARE OF THE CHILD.

"THE Principles of Antenatal and Postnatal Child Hygiene," by Dr. W. M. Feldman, is an encyclopædic tome of some seven hundred pages[1]; it is a companion volume to "The Principles of Antenatal and Postnatal Child Physiology" by the same author. The book deals with child hygiene at every period, antenatal (including anteconceptional) intranatal and postnatal up to puberty, in other words it deals with the young life before it begins, while it is in the making and in all stages of infancy and childhood.

As in other writings of Dr. Feldman the historical aspect has due emphasis, the influence of religion and race is considered and so perspective is given to the modern developments of child welfare. Another of the author's attributes is his skill in mathematics and a chapter is devoted to biometrics which should be valued by those dealing with statistics.

Every chapter throughout the book is full of information culled from many sources and methods in use not only in England, but in foreign countries, are quoted and thus interesting comparisons are afforded. For instance, birth control is discussed, the arguments for its widespread adoption are canvassed and the author's opinion is divined as being in opposition to the practice. Its legalization and the facilities offered in Soviet Russia and Armenia are mentioned, no doubt a providential arrangement to limit undue growth of these nations, but better avoided by our own.

Maternal mortality and infant mortality are discussed from each point of view, historical, statistical, preventive and a wealth of information is provided. Heredity, eugenics and inevitably the subject of mental deficiency pass in review and then the scene changes to the physiology and care of fœtus, expectant mother, baby *et cetera* and so to breast feeding and artificial feeding of infants.

Diets are given for mothers, babies and older children and the thousand and one details which must be considered by those dealing with such subjects are clearly set forth. While all will not agree with some of the diets given, for example, that for the expectant mothers on page 270, yet in the main they represent the modern view of dietetics. Heliotherapy, the care of the sense organs of the premature infant, the prevention of infectious diseases and many other equally important subjects are included in this volume which is certainly a very interesting and valuable book of reference. The historical setting, the wealth of detail and the scientific attitude of the author to these questions should make it of great value to all medical practitioners, students and welfare workers of the English-speaking world.

The book is well printed and the illustrations are numerous. Particularly interesting are the photographs of famous workers for the welfare of children, a large number of which is included. The bibliography is exhaustive and in itself a valuable contribution to the subject. Dr. Feldman is to be congratulated on the result of his labours which must have been colossal.

[1] "The Principles of Ante-Natal and Post-Natal Child Hygiene," by W. M. Feldman, M.D., B.S., M.R.C.P. (London), F.R.S. (Edinburgh); 1927. London: John Bale, Sons and Danielsson, Limited. Royal 8vo., pp. 765, with illustrations. Price: 25s. net.

PASSIVE MOVEMENT IN ARTHRITIC CONDITIONS.

IN "Treatment by Manipulation,"[1] A. G. Timbrell Fisher attempts to put on a rational basis the treatment of many diseases and injuries which frequently confront the practitioner.

His theme is that improved nutrition and resistance to disease must follow if adequate function is secured and he never loses sight of this fundamental and generally recognized fact. The logical outcome of an acceptance of this doctrine is that the practitioner who would acquire skill in manipulation of injured or diseased joints, muscles and fibrous tissues, must be conversant with the detailed anatomy, physiology and pathology of these structures.

The author maintains that rest may be a pernicious influence if it is misapplied and he joins issue with those who unduly prolong the period of rest for the common sprains, after operations on joints and in many cases of chronic arthritis. "A too rigid observance of the doctrine of rest leads to two very serious complications—wasting of muscles and adhesions." At the same time he maintains that the normal range of movements of a given joint must be carefully borne in mind and the reader is furnished with complete descriptions of these.

Before a diseased joint is manipulated the presence of tuberculosis must be carefully excluded by X rays and other available means of diagnosis, but it is illogical to manipulate a joint for the purpose of increasing movement and then immobilize it for a long period in plaster of Paris.

Eburnation and sclerosis represent Nature's efforts to provide a new joint surface and they should be assisted by movement, particularly in the more chronic forms of arthritis associated with lipping of the articular surface. The argument is supported by interesting case reports which are neither too numerous nor tedious. Various dislocations and subluxations are dealt with and a clear description of the pathological basis of signs and symptoms assists the reader in applying the principles of treatment outlined.

The section on functional disabilities has apparently been sacrificed to the demands of space. Greater detail in this section would assist the reader in obeying the injunction to establish all treatment on the foundation of an accurate diagnosis.

The letterpress is good with the exception of two obvious misprints, "osteometies" on page 35 and "rise give" on page 49 and the illustrations are adequate for the subject.

The book should appeal to all those who wish to make their treatment of such conditions less empirical than that adopted by the osteopaths and similar cults, and is sufficiently brief to satisfy the demands of the busy general practitioner.

[1] "Treatment by Manipulation: A Practical Handbook for the Practitioner and Student," by A. G. Timbrell Fisher, M.C., F.R.C.S. (England); 1928. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 212, with illustrations. Price: 9s. net.

The Medical Journal of Australia

SATURDAY, JUNE 23, 1928.

The Journal of the College of Surgeons of Australasia.

THE Council of the College of Surgeons of Australasia has determined to publish a journal. The first number will probably be issued in July of this year. This decision has been formed after much deliberation. It was originally suggested that the Australasian Medical Publishing Company, Limited, should publish the journal with an editorial Committee appointed by the College of Surgeons. The directors of the company, however, were not prepared under the existing conditions to accept the financial risk. The proposal was therefore postponed. During the course of last year the Council of the College found that it needed a medium for approaching its fellows and members. One of the objects of its foundation is to advance the science and art of surgery and to encourage members of the medical profession to become efficient surgeons. It was thought that the publication of original articles of didactic value, the records of research by competent observers and critical discussions on work carried out in other parts of the world would be of great benefit not only to its own fellows and members, but also to the whole of the practising members of the medical profession in the Commonwealth of Australia and the Dominion of New Zealand. Hitherto this function has been performed by THE MEDICAL JOURNAL OF AUSTRALIA. It is thought that the appearance of *The Journal of the College of Surgeons of Australasia* would not impair the usefulness of this journal and that the two journals could be of benefit to the medical profession with ample scope for the development of each. The Australasian Medical Publishing Company, Limited, therefore, has undertaken to print *The Journal of the College of Surgeons of Australasia*. It will remain the property of the College, will be produced by an editorial committee, with Dr. Mervyn Archdall as editor and will be circulated among the fellows and members of the College and to subscribers. We welcome the new journal and wish it a prosperous and useful career.

It is anticipated that many medical practitioners will wish to read the new journal. Some information concerning it may therefore be welcomed. The first number will contain approximately 120 pages. The journal will be crown quarto size, that is ten inches by seven and a half inches or as we should put it twenty-five and a half centimetres by nineteen centimetres. The contents will include the transactions of the first annual meeting of the College, held at Canberra on March 30 and 31 and April 2, 1928. It will be remembered that in addition to the papers on surgical subjects, there were some on the history of the colleges of surgeons and there was also a debate on the hospital question. All these will make good reading. It is hoped that those responsible for the journal will restrict the contents of subsequent issues to purely surgical matter and will not lend its pages to questions of medico-politics, a subject which does not come within the survey of the College and for the handling of which the College has no machinery.

The price will be ten shillings and sixpence for a single number. It has not been decided how many numbers will be produced in each year, but we understand that there will probably be three. The price is not high. A single copy of *The British Journal of Surgery* is sold for twelve shillings and sixpence. The average number of pages per issue is about 180. The size of the page is the same as that proposed for *The Journal of the College of Surgeons of Australasia*. The British and American journals are profusely illustrated; the Australasian journal is unlikely to lag behind them in this respect. Fuller particulars will be published in the near future. In the meantime those who are anxious to secure copies of the first number, are advised to send their orders either to the Honorary Secretary of the College of Surgeons of Australasia, Collins Street, Melbourne, or to the Manager, The Australasian Medical Publishing Company, Limited, The Printing House, Seamer Street, Glebe.

Current Comment.

NARCOLEPSY.

THE clinical condition known as narcolepsy was first described by Gélineau in 1880. Since then many cases have been reported and there has been much discussion about the true nature of the syndrome and about the place which it should occupy in the classification of diseases. In 1926 Adie wrote a long article on the subject and described idiopathic narcolepsy as a "disease *sui generis*." An abstract of this paper was published in THE MEDICAL JOURNAL OF AUSTRALIA of March 19, 1927. In expressing the view that Gélineau's narcolepsy is a disease *sui generis*, Adie stated that the condition is not very rare and that it is commonly mistaken for epilepsy or hysteria, although it is distinct from both. The characteristic symptoms, sudden irresistible attacks of sleep with cataplexy or inability to move a muscle or utter a word, have been described in this journal by Willis and by Bostock in the issue of August 20, 1927. The determination of the nature of this condition and of its relationship with other pathological states arising in the nervous system is of such importance that careful consideration should be paid to a recent communication by S. A. Kinnier Wilson.[1]

Wilson bases his findings on observations made of four patients and on thirty-nine whose histories have been recorded by others. He states that from the point of view of nosology it is unscientific and reactionary to allude to any of the disorders of sleep as diseases. It may or may not be regarded as established that there exists a special clinical type of sleep disorder, a clinical syndrome corresponding to Gélineau's original description of a "neurosis" to which he gave the name narcolepsy, but "at the most it remains only a clinical combination." The fact that narcolepsy has been reported as a sequel to epidemic encephalitis has in Wilson's opinion added to the impracticability of its isolation as a *morbus sui generis*.

The first case described by Wilson is that of a man, aged twenty-four years, who had suffered for three years from continually recurring diurnal attacks of sleep. These seizures were associated with attacks of tonelessness, "cataplexy," in the limbs which occurred under the influence of emotion —whenever he laughed heartily he experienced a giving way of the knees. He also had frequent attacks of diplopia. The second patient was a man, aged twenty-six years. He had a brother who suffered from a condition described as epilepsy. He suffered from typical diurnal attacks of sleep and from numerous attacks of cataplexy. These attacks of tonelessness arose in two distinct ways—either under the influence of an emotion or because he was trying to avoid an oncoming attack of sleep. In regard to the first of these, any excitement sufficed. Laughter or temper produced an attack and the tendency to tonelessness was always increased if the patient thought anyone was looking at him. Wilson had an opportunity of examining the patient's deep reflexes while the latter was overcome by an attack of cataplexy. The muscles were atonic and flaccid, the pupils reacted slightly but definitely to light, the knee jerks were completely abolished on both sides and the plantar reflex was extensor in type. The patient was conscious the whole time. The third patient was a man who had suffered for twenty years. In his case the attacks were of three different types. He had typical short diurnal sleep seizures. He experienced momentary loss of power in the limbs under the influence of any excitement, such as laughter or rage. He passed into trance-like states of absolute paralysis during which no movement was possible; consciousness was unimpaired and the condition was not brought on by emotion, but usually occurred when the patient was resting or sitting quietly. The fourth patient was a schoolboy aged eighteen years. Two or three months before the onset of the condition he suffered from an indefinite illness which was diagnosed as epidemic encephalitis. He suffered from short diurnal attacks of sleep and also from attacks of loss of power in the limbs under the influence of emotional stimuli. The attacks of sleep were usually quite brief and on several occasions it was more akin to a state of day dreaming. The point to be noted about these four patients is the variation in severity of symptoms. All had typical diurnal attacks of sleep. In the fourth patient they resembled reveries, though to all appearances the boy was asleep. In the others the sleep attack was more typical. The relationship between the cataplexy and sleep attacks is evident, particularly in the second patient whose cataplexy supervened when he struggled against the desire to sleep. Further, the trance-like state of "absolute paralysis" in the third patient complicates the picture. It is stated that the patient had a paralysing feeling and that his limbs relaxed. It is unfortunate that no account of physical examination during this stage is recorded. The patient was apparently in a more advanced stage of "tonelessness." The description is not unlike that of a patient in catalepsy.

The classification of any disease depends primarily on a knowledge of the pathogenesis. In the present state of knowledge no scientific classification of narcolepsy is possible and it is necessary to fall back on an empirical grouping from the clinical standpoint. In Wilson's opinion a distinction into "idiopathic" and "symptomatic" varieties is not calculated to further knowledge for the reason that idiopathic narcolepsy cannot be causeless and cases analogous to it in every respect have been seen to follow an attack of epidemic encephalitis. He offers a purely tentative division with two main classes and claims that, although artificial, it may prove clinically convenient. The first class comprises recurring diurnal attacks of sleep and the second includes sleep attacks of longer duration, day and night sleeps running into each other and continuous sleep. The first class is further subdivided into sleep associated with attacks of tone-

[1] *Brain*, March, 1928.

lessness (cataplexy) and those in which toneless attacks do not occur. Wilson considers this grouping and discusses the cases reported in the literature from this point of view. Thirty-nine cases gathered from the literature and his own four cases fall into the first of these latter subdivisions. The cases of Willis and Bostock, previously mentioned, also fall into this subdivision. The ratio of male to female patients among those referred to by Wilson is over six to one. In discussing the second subdivision of the first group—diurnal attacks of sleep without "toneless" attacks—Wilson points out that sleep attacks associated with certain forms of drug taking and with certain cerebral tumours really come into this division. The chief impression gathered from a survey of this large class is that as far as recurring irresistible diurnal attacks of sleep are concerned, there is no affinity whatever between them and any one specific invariable form of disease. Seeing that attacks of sleep occur as a monosymptomatic variety of narcolepsy, the question arises as to whether the loss of tone may be the only symptom of the condition. As far as Wilson can discover, there is no record of attacks of loss of power or tone occurring through influence of the emotions. He describes the history of one patient, however, whom he regards as suffering from "larval or incomplete" variety of narcolepsy. He assigns this case to an abortive or incomplete class in the general group and holds that only the affective or toneless attacks have passed into a characteristic stage.

One point of great importance is the fact that some patients have only the appearance of sleep; they are in reality aware of their surroundings, but are not able to move a single muscle or articulate a single word. In other words, the condition bears only an apparent resemblance to sleep and is transitional to the cataplectic class of seizure. There are differences between the sleep of a narcoleptic patient and that of a normal person and Wilson refers to work by Richter who described changes in the electrical resistance of the body during sleep. He thinks that work along these lines may throw light on the differences between normal sleep and pathological conditions closely resembling it. He also discusses the toneless attacks at some length and points out that it is erroneous to suppose that the two types of attack are essentially dissimlar and are correlated only through their occurrence in one and the same patient. The transitions and substitutions noted clinically are sufficient evidence of this.

One of the most important aspects of the subject of narcolepsy is its relationship to epilepsy. This has been mentioned on a previous occasion in these columns. If it be assumed that epilepsy arises as a result of the irritant action of certain chemical substances on nerve cells which are hyperirritable, it is but natural to regard narcolepsy as having a somewhat similar origin. Just as epilepsy is really a symptom and not a disease, so must narcolepsy be regarded. Similarly, as most neurologists follow the teaching of Hughlings Jackson and speak of "the epilepsies," Wilson is justified in his contention that the syndrome under discussion should be referred to as "the narcolepsies" rather than as narcolepsy.

Wilson attacks those who object to the idea that there is any association between epilepsy and narcolepsy. He states that the immediate and obvious answer is to ask what they mean by epilepsy. Until a definition of epilepsy is furnished by these objectors, it is useless to say that narcolepsy has nothing to do with it. Wilson takes the view that the unknown diseased condition is the cause of epilepsy and that the "epilepsy" is a symptom pure and simple and, as such, strictly physiological. So he argues that any alleged contrast between the narcolepsies and the epilepsies is merely a contrast between symptoms and leaves untouched the problem of whether or not there is a similar method of production behind each. The general resemblance between the two classes of symptoms must be admitted. It is impossible to overlook the analogies which consist in periodical recurrence of attack, abrupt onset with or without aura, loss or diminution of consciousness and relatively brief duration. He states that if behind the ordinarily great dissimilarity of the external manifestations there is reason to postulate the action of not dissimilar mechanisms, it becomes unscientific to argue for fundamental differences between them from a nosological point of view. He analyses some of the views expressed by Adie. The latter holds that the most important feature of idiopathic epilepsy is that the attacks come on without apparent cause, whereas in narcolepsy the cataplectic attacks are "reactive," in other words there is a definite exciting cause, emotion. He further holds that in genuine epilepsy the attacks are never reactive and that this feature alone is sufficient to show that the two conditions are fundamentally distinct. It has, however, been pointed out by Goldflam and Somer that cataplectic attacks may develope spontaneously and the trance-like states of the third and the fifth patients described by Wilson occurred without any emotional antecedent. Wilson also states that the view that idiopathic epilepsy is never reactive is contradicted by familiar experience. In his opinion the most important positive argument is the fact that epilepsy and narcolepsy have appeared in the same individual. Wilson concludes that it would be a mistake both to ignore the similarity and to lay stress on the differences between the two conditions; the correct attitude is to suspend judgement and to await further contributions to knowledge. At the same time he believes that the considerations put forward by him make out a *prima facie* case for the possibility of some of the narcolepsies and of the epilepsies having a common origin. His final statement is that it is useless to force cases of narcolepsy into the category which seems suited to them unless they fit comfortably. This is counsel of perfection, for until more is known, there will certainly be many misfits. In the meanwhile accurate clinical observation must be carried out by every practitioner seeing a patient who may reasonably be suspected of suffering from some form of narcolepsy.

Abstracts from Current Medical Literature.

DERMATOLOGY.

The Causation and Treatment of Pruritus.

L. ARZT (*Wiener Medizinische Wochenschrift*, January 1, 1928) discusses the value of modern methods in the treatment of pruritus. In the treatment of scabies he recommends "Mitigal"—a colloidal preparation of sulphur which does not soil clothing and has few ill effects. The oil is rubbed in four times within an hour. The next day a bath is taken and Lassar's paste is used for any resulting skin irritation. Another useful preparation is "Catamin," which contains 5% sulphur and 10% zinc ointment. Pediculosis is best treated with preparations of copper sulphate suspended in liquid paraffin. Eczema is the principal non-parasitic cause of pruritus. The acute forms are best treated with intravenous injections of ten cubic centimetres of a 10% aqueous solution of sodium bromide every three or four days. Calcium therapy is indicated for more chronic conditions either in tablet form or as injections. Urticaria should be treated with combinations of calcium or strontium chloride with intravenous injections of urea in doses of five to ten cubic centimetres. Venesection with reinjection of the serum (two to five cubic centimetres) has also proved of value. Pruritus is also caused by *lichen planus*. The treatment has usually been carried out with arsenical preparations. On the assumption of an infectious origin milk injections have also proved efficacious. Definite pruritus has also followed "Salvarsan" injections. This is overcome by intravenous injections of sodium thio-sulphate, 0·3, 0·45, 0·6 and 0·9 gramme in ten cubic centimetres of water on successive days. Senile pruritus, frequently of unknown causation, is relieved by intravenous injections of 0·5 to 1 cubic centimetre of a 1% aqueous solution of sodium silicate. About eighteen injections every second day are required to effect a cure. This treatment is particularly useful in scleroderma.

The Use of the "Grenz" (Infra-Röntgen) Rays in Dermatological Conditions.

J. J. ELLER AND G. BUCKY (*Archives of Dermatology and Syphilology*, February, 1928) describe the result of five years of investigation into the physical and biological properties of a range of wave length between the ultra-violet and the Röntgen rays. It was thought that the effects of rays of both these types might be combined and the authors claim that the results more or less confirm the correctness of this assumption. The term *Grenzstrahlen* or borderline rays was applied to these waves which had an average length of two Ångström units. Their penetration is small and two millimetres of water will absorb 90% of them. By using the "*Grenz*" rays it is held that less damage is done to the deeper tissues, as practically all of the absorption takes place in the skin. A special apparatus producing a low tension between 5,000 and 9,000 volts is necessary. This voltage is equivalent to 1.06 to 1·9 millimetres between spheres of two centimetres diameter. The tube is of the heated filament type. No epilation of hair occurs. A dose that was necessary to produce a mild erythema in twelve to twenty-four hours was regarded as a unit. The dose can be repeated in two weeks. Most of the diseases required two unit doses for results. Atrophy or telangiectisis has not been observed.

Keratoderma Blennorrhagicum.

A. ROSTENBERG AND H. SILVER (*Archives of Dermatology and Syphilology*, December, 1927) describe a case of *keratoderma blennorrhagicum* in a man aged thirty-two years. They also review seventy previous cases from the literature and discuss the pathogenicity of the disease. The authors come to the conclusion that *keratoderma blennorrhagicum* is not a true keratosis, but an inflammatory process beginning with a vesicle which dries up and forms a keratotic lesion. The patient described by them had a generalized eruption with a large number of rupial lesions. They hold that gonorrhœa is not alone responsible for the syndrome and suggest a name which, they claim, conveys a true clinical picture, *dermatitis rupoides arthropatica*.

Shallium in Ringworm of the Scalp in Children.

B. F. FELDEN (*Archives of Dermatology and Syphilology*, February, 1928) discusses the origin of the use of shallium acetate in producing alopecia and gives a report on the administration of this drug in a series of forty-seven cases of feeble-minded children in a New York hospital. Forty-five of the children were between the ages of two and thirteen years. The dose was eight milligrammes per kilogram of the body weight. The hair commences to regrow in from three to four weeks. Local treatment was kept up the whole time. Of the forty-seven patients treated, thirty-six were cured after one medication. Toxic symptoms were noted in only one patient and this was a girl approaching puberty. The author states that this could have been avoided by a stricter adherence to the rule that shallium should be given only to children before puberty. Expilation is complete only in about 50% of cases and has to be assisted by adhesive strips. Local treatment by painting the scalp with 5% tincture of iodine and the daily application of a 10% sulphur ointment should be continued throughout. Patients with a pronounced discrepancy between weight and age are unfit for pure shallium treatment, the combined method, consisting of one-half or two-thirds of the dosage of both X rays and shallium, offers the best outlook. Shallium is strictly to be avoided in adults and in children approaching puberty because of its serious toxicity. It should be taken on an empty stomach dissolved in sugar water and a second dose should not be given until two months have elapsed.

The Pityrosporon of Malassez.

J. M. H. MACLEOD AND G. B. DOWLING (*The British Journal of Dermatology and Syphilis*, April, 1928) suggest that the term "spore of Malassez" should be used to describe the "bottle bacillus" of Sabouraud. A useful method of staining is by putting a drop of undiluted Giemsa stain at the edge of the cover slip. The organism is pleomorphic, generally rounded or flask-shaped and as a rule occurs in groups or chains. It can be grown on Sabouraud's maltose agar or on acid glycerine agar and occurs aerobically at 25° C. With 1% sugar solution and 1% culture in peptone, in galactose, maltose and glucose the medium becomes acid, with slight gas formation in the two latter. In saccharose, inulin, dulcite, raffinose, lactose and mannite the authors found no change. The fungus belongs to the yeast-like group of *bosporaceæ*. In order to try to establish a definite relationship between the organism and the seborrhœic lesions three methods of actual inoculation were tried on the human subject: (i) a suspension was rubbed into the skin, (ii) it was injected intradermally, (iii) it was implanted after scarification. With the last two methods a rash, identical with seborrhœic dermatitis, was always reproduced. Two groups of patients were inoculated, those already with seborrhœic dermatitis and those without and all reacted. These observations and experiments appear to prove that seborrhœic dermatitis is a pure fungus infection caused by the pityrosporon of Malassez.

Fluorescence, Particularly in Dermatology.

H. GOODMAN (*The British Journal of Dermatology and Syphilis*, March, 1928) makes some observations and forecasts on the use of fluorescence in dermatology. He has been in possession of a filter which allows the passage of ultra-violet rays down to 2,400 Ångström units and yet bars the visible radiation except for a small amount of visible violet light. By this means he has studied the fluorescent appearance of many skin conditions. Lesions such as warts, epidermal scales, fringes of scaly lesions give the characteristic fluorescence of the horny layer composing them as evidenced in the teeth, nails and normal palms. Fluorescence in the hair is in an inverse ratio to its pigment content. Inflammatory reactions of the skin surface present a dark appearance, as also do angiomata and pigmented areas. The skin of coloured people has a fluorescence

quite different to that of pigmented lesions in white persons. Areas, radiated by the mercury vapour lamp, are indicated shortly after exposure hours before an erythema appears in both coloured and white skinned people.

Urticaria Pigmentosa.

J. F. FRASER AND M. N. RICHTER (*Archives of Dermatology and Syphilology*, April, 1928) describe the history and microscopical findings in two cases of *urticaria pigmentosa* of "adult" type. In one case the disease did not appear until the patient was thirty years of age and was moreover complicated by the presence of a positive response to the Wassermann test. The rash, however, did not disappear with antisyphilitic treatment. The microscopical changes in the "adult" type were essentially the same as in other cases and consisted mainly of: (i) pathological mast cell accumulation in the corium, (ii) rarefaction of the collagen bundles with a tendency to fraying and rupture of the elastin and (iii) the presence of pigment granules (melanin) in the epidermis and also in deeper areas. The mast cells are held to be morphologically and genetically different to the mast cells of the blood, the nucleus being single and rounded or oval, not polymorphous. It is claimed that they arise locally from connective tissue cells or lymphocytes. No therapeutic measures were found to be of any use.

UROLOGY.

The Identification of the Tubercle Bacillus in the Urine.

H. S. JACK AND M. A. MUNCH (*Journal of Urology*, December, 1927) have made a comparative study of the various centrifugalization methods used in seeking for tubercle bacilli in the urine. They conclude that the best results are obtained when the specimen is spun for a period of not less than fifteen minutes at a speed of about 2,300 revolutions per minute. A period longer than fifteen minutes is apparently unnecessary. This is an important factor, as well as centrifugal force, in carrying down tubercle bacilli. If a part of the pus is removed in order to procure better smears, the pus so removed should be examined for tubercle bacilli as well as the final sediment. Simple centrifugalization is equal to any other method, including those in which chemical reagents are used, and is better than most of them.

The Diagnosis of Horseshoe Kidney.

D. N. EISENDRATH, D. F. RUDNICK AND W. BAKER (*Journal of Urology*, August, 1927) report on three cases of horseshoe kidney diagnosed before operation. Certain clinical findings combined with suggestive appearances of the renal shadows in the plain skiagrams may make the surgeon suspicious of the renal condition, but the use of pyelography has made preoperative diagnosis possible, whereas until recent years the diagnosis was never made until operation was undertaken. The pelvis and calyces are usually lower than normal on both sides and are nearer the middle line. Moreover, as with certain other congenital abnormalities, the ureter is inserted on the lateral side of the pelvis. If the pelvis is lying transversely, the ureter is inserted into its lower border. Correspondingly the calyces are directed medially or upwards and not laterally, as is normal. As they approach their lateral insertion into the pelves, the upper parts of the ureters generally deviate laterally; this is a very characteristic feature. In all three cases the patient's symptoms were due to calculi in one or both halves of the horseshoe kidney. As routine pyelography was used, the actual diagnosis of the congenital abnormality was made before it was decided to perform an operation. At operation the question of hemi-nephrectomy has often to be considered and this amputation may be done with the aid of special mattress suturing, provided the remaining half of the kidney shows good efficiency.

Gradual Decompression of the Bladder.

W. W. SCOTT (*Journal of Urology*, January, 1928) advocates the insertion of an ordinary Number 5 French ureteric catheter into the bladder *per urethram* to secure gradual decompression of the bladder in chronic retention of urine. After studying the question carefully he considers that no advantage is to be gained in using complicated apparatus designed to spread the emptying of the bladder over a period of several days; in fact, a relatively rapid method gives better results. The drainage through the small ureteric catheter is regular and sufficiently slow, taking several hours to empty the bladder.

The Inlying Ureteric Catheter.

D. N. EISENDRATH (*The Journal of the American Medical Association*, December 24, 1927) makes a plea for the more widespread use of the inlying ureteric catheter in pyelonephritis and certain other urological conditions. Its field of application includes the treatment of acute and chronic pyelonephritis, the relief of anuria of obstructive origin, the relief of severe pain in renal and ureteric calculus or in kinking of the ureter in a kidney which has undergone ptosis, the deviation of the urine after vesico-vaginal fistula operations and the "splinting" of the ureter when it is injured during hysterectomy. The catheter may be left in on one or both sides for periods up to several weeks, if necessary. A small (Number 5 French) catheter is preferable, since it permits drainage alongside as well as through its lumen. Lavage of the renal pelvis is rarely necessary, except to dislodge particles blocking the catheter. If there is a tendency for the catheter to be expelled, a small catheter should be tied in the urethra to keep the bladder empty. If fever persists in spite of the use of the inlying catheter in both acute and subacute cases of pyelonephritis, consideration should be given to the possibility of an extension of the infection, either to the perirenal tissues or of such a severe degree of parenchymal involvement as to render surgical operation necessary.

The Bladder in Tabes Dorsalis.

E. D'ARCY MCCREA (*Irish Journal of Medical Science*, November, 1927) has made careful clinical observations in thirty cases of tabes affecting the bladder. He divides the patients into two main classes: (i) the incontinent, in whom sensation is cut off from the higher centres, and (ii) the paretic whose sensory loss, though present, is not so evident and is concealed by the interference with the reflex function of the viscus. A third group, in whom the symptoms are of a truly mixed type, exists, but is rare. In the incontinent type the main symptoms are similar to those accompanying a lesion of the sensory pathway of the spinal cord and are characterized by a loss of the mucosal and muscle senses of the posterior portion of the urethra. These senses are transmitted by the pudic nerves which are somatic. In the paretic type the main symptoms are due to lesions of the afferent fibres running in the pelvic nerves which are autonomic.

Second Removal of the Prostate in Carcinoma.

A. L. CHUTE (*The Boston Medical and Surgical Journal*, December 29, 1927) advocates a second removal of prostatic tissue by the perineal route when symptoms of obstruction have arisen after removal of the prostate for carcinoma. This is purely a palliative measure and is designed simply to relieve the patient. Back-pressure on the kidneys, arising from recurrent malignant obstruction, presents the same dangers as urinary back-pressure in general. In removing the recurrent malignant tissue, dissection with some moderately sharp instrument is necessary, as enucleation by the finger is usually impossible.

Infarction of the Testicle.

S. LUBASCH (*Journal of Urology*, October, 1927) considers that infarction of the testicle does occur and should be thought of as an entity. The testicle, having a terminal circulation, should be placed in the same category as the spleen, kidney *et cetera*. The ætiology, though still obscure, may be classified as consisting of predisposing and exciting factors. The former comprise any condition that would cause embolism. The latter include a temporary twisting of the spermatic cord, resulting in stasis of one of the arterioles. Infarction should be suspected when symptoms of torsion recur after abatement. The treatment is orchidectomy.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society Hall, East Melbourne, on May 2, 1928, DR. J. NEWMAN MORRIS, the President, in the chair.

Common Cardiac Problems.

DR. M. D. SILBERBERG read a paper entitled: "Common Cardiac Problems: Syncope, Cardiac Neurosis, Præcordial Pain" (see page 764).

DR. ALBERT WEIGALL asked Dr. Silberberg's opinion of the so-called tobacco heart. This seemed to occur in two types of patient: the young man who smoked too many cigarettes and complained of shortness of breath on exertion and exhibited a rapid or so-called nervous heart action without much alteration in blood pressure, and the older man who perhaps smoked cigars or pipe tobacco, six or eight ounces a week, and tended to have a low systolic blood pressure and a poor pulse pressure. Their symptoms improved if their tobacco were cut down.

DR. A. V. M. ANDERSON congratulated Dr. Silberberg on his appointment to the senior staff of the Alfred Hospital. This should be to the advantage of the patients and students. He referred to the fact that in consulting practice many patients appeared to be unduly alarmed as to the seriousness of a heart condition and for this the medical adviser of the patient was sometimes responsible. Richard Cabot in his work entitled "Facts on the Heart," stated that the most important point of all was to know that most "heart disease was imaginary" and this dictum was often the most comforting to a patient with no signs of organic disease. He preferred the term premature systole to extra systole as being more accurate. Stress had been laid recently on the importance of "reflexigenous zones" in the production of *angina pectoris* and Dr. Verdon in his book had stated his opinion that spasm of the œsophageal end of the stomach was not uncommonly the exciting cause of an attack. Dr. Anderson believed that infradiaphragmatic conditions and particularly gall stone diseases were not an infrequent precursor of anginal attacks and instanced a case in which the connexion between the two conditions seemed to be close. In any case when presternal or substernal pain on exertion existed he thought that the pain should be regarded as possibly anginal.

DR. L. S. LATHAM said that he had enjoyed Dr. Silberberg's clear, succinct and scholarly description of his clinical cases. These descriptions had been so excellent that very little could be added. He agreed that fear of death dominated the popular attitude towards cardiac disease. It would be helpful to be able definitely to group those conditions. Many practitioners had encountered an unexpected fatal termination in a case which had been given a more favourable prognosis. It was difficult to measure myocardial degeneration with certainty, even with elaborate methods. It was important to note the relation of symptoms to exertion, especially in the first attack, as emphasized by Dr. Silberberg. It was well to remember that a mild attack might be an early or minor evidence of a major type of lesion which might prove fatal. In his experience the cardiophobe seemed less common since the Great War, probably due to the fact that the public recognized that in a large proportion of cases the various heart disabilities from which returned soldiers suffered did not prove fatal.

DR. W. OSTERMEYER expressed appreciation of Dr. Silberberg's paper and agreement with his conclusions which represented a considerable amount of important clinical applied research. It was important to note that the momentary syncope exhibited by some patients was due to premature systoles and not to myocardial degeneration. Dr. Ostermeyer related some intimate personal experiences which supported the dental toxic origin of one large class of premature systole, temporary in character. He agreed that the cardiophobe was less commonly met with than formerly. It was over thirty years since Gowers had strongly brought out the truth that the clinical picture of organic nervous disease might be painted on a functional background and *vice versa*. Dr. Silberberg had well emphasized the fact that cardiac disorders, too, might be a mixture of organic and functional types in varied proportion.

DR. LESLIE E. HURLEY also expressed appreciation of the paper and went on to discuss certain points that had helped him in the diagnosis of organic *angina pectoris* from so-called pseudo angina. The condition of the patient between attacks was an important guide. In cases of toxic or pseudo angina the patient was frequently out of sorts between attacks, the history was vague and the attacks might last half an hour or more. The patient with organic angina usually felt well between attacks, the attack was a matter of seconds or minutes and the history was usually given in a definite and clear-cut manner. In true angina the administration of a vasodilator to open up the peripheral circulation usually relieved the attack. When cardiac failure occurred with œdema and perhaps tricuspid regurgitation, angina usually disappeared, while the recovery of cardiac efficiency might lead to the return of angina. These facts suggested that the pain in angina might be related to increased cardiac or aortic tension. The operation of cardio-aortic sympathectomy could not be justified if angina were looked upon as a protective mechanism, but only if it were considered that the actual attacks were dangerous in impairing myocardial efficiency, as held by Clifford Albutt. He had seen one elderly lady suffering from syncopal attacks. On examining her during an attack he had found the pulse rate at the wrist 34 and at the apex beat 68 in the minute. The condition had been due to a series of alternate extra systoles and he had seen several such cases.

Concerning the relationship between gall bladder disease and heart disease, he had a patient with auricular fibrillation and pain at the right costal margin which was thought to be due to congestion of the liver. This patient later had had typical biliary colic. Gall stones had been removed and the auricular fibrillation ceased.

A young man, aged twenty-eight years, in his out-patient clinic had had an irregular pulse for six or seven years which he had discovered himself but which had not caused any unpleasant symptoms. He had been refused a permanent appointment in the Railways Department because of it and his *fiancée* had refused to marry him for the same reason. The electrocardiograph had confirmed the clinical diagnosis of auricular fibrillation. He had had a suggestion of exophthalmos, but the basal metabolic rate had been normal. He had been given quinidine in usual doses with a rapid response. The pulse had become regular and had remained so for twelve months. He was still taking quinidine in doses of 0·2 gramme daily and was doing his usual work as a fitter.

Dr. Hurley asked Dr. Silberberg how long he would advise the quinidine to be continued after the pulse became regular. In some American clinics quinidine was discontinued as soon as the pulse became regular. Was this wise? He asked if Dr. Silberberg had seen any case of coronary thrombosis in which the patient did not complain of pain. The speaker had seen patients with arteriosclerosis and cardiovascular degeneration undergo a sudden alteration in their general condition with gross cardiac failure, but no anginal pain. Autopsy in some of these cases had revealed coronary thrombosis.

A recent classification of types of coronary thrombosis had grouped such cases into: (i) those with pain and the usual typical features, (ii) those without pain but with a sudden development of cardiac failure, (iii) those with gradually progressing myocardial failure but with no dramatic alteration in the clinical picture at the onset of the thrombosis.

DR. J. NEWMAN MORRIS, the President, expressed his great appreciation of Dr. Silberberg's paper. He was interested in the surgical aspect of the problems of cardiac disease. Should the thyreoid gland be removed in cases of irritable heart with increased metabolic rate? Some surgeons did so with varying results. The speaker had had a patient, a man with stare and tremor, but no thyreoid enlargement. He had become suddenly ill with a pulse rate of one

hundred and thirty beats per minute and over. He had worked hard for thirty years without sick leave.

He had been treated by various physicians without benefit with Lugol's solution, digitalis *et cetera*. Eighteen months later Dr. Morris had removed this man's thyreoid gland in two stages. The pulse had become normal and the tremor had disappeared after the removal of one lobe, but he had relapsed two months later and the other lobe had been removed. He again recovered, but in four or five months his symptoms had partly returned, but had not been nearly as severe as before. He had returned to his work. Dr. Morris suggested that this second relapse had been due to the functional overaction of remnants of thyreoid tissue left at operation.

Dr. Silberberg in reply referred to Dr. Anderson's vast clinical experience, thanked him for his congratulations and said that he felt that he could not worthily fill Dr. Anderson's place on the staff.

Replying to Dr. Weigall, Dr. Silberberg said that tobacco acted directly on the sympathetic nervous system and indirectly on the heart. The excessive use of cigars would be likely to cause more real harm than cigarettes as they were "a heavier and longer smoke." Toxic foci were real but elusive factors in some cases of the type described by Dr. Weigall, quite apart from smoking. In Cabot's opinion symptoms of cardiac distress were due to cardiac neuroses rather than to organic disease in at least 10% of cases.

Referring to Verdon's œsophageal gastrospasm, the speaker said that the *triangularis substernalis* muscle might be the seat of the spasm in some instances. Cardiology was not a narrow specialty and it was necessary to investigate the whole of the nervous and organic systems and also to take into account the psychological condition of the patient. Dr. Silberberg made a plea for the more systematic recording of clinical findings by hospital staffs and the more thorough correlation of such records for purposes of clinical research. At present there were general impressions, but no satisfactory records.

The relation of syncope to extra systoles was real, but not common. Coupled beats occurring during the administration of digitalis were a danger sign and possibly indicated impending ventricular fibrillation. It was common to find organic lesions associated with a superimposed functional syndrome.

The relation of angina to low blood pressure was puzzling; patients with low blood pressure obtained little relief from vasodilators which might cause fainting. Lowering of pressure was not the essential factor in the relief of all patients with angina and cardiac failure did not always cause cessation of anginal pain. Such relief, when it occurred, might be due to the cessation of activity. Pneumonia might reduce the blood pressure in some instances and in one patient the pressure had remained down for twelve months, but the patient had eventually died from angina. This lowering of pressure might be associated with the liberation in the system of histamine and it was suggested that histamine might prove useful therapeutically. The relationship of gall bladder disease to fibrillation was comparable to the disappearance of fibrillation after thyreoidectomy.

With regard to the continued administration of quinidine, he thought that each patient should be considered individually and the drug continued till the heart action became regular, but he did not see any reason for continuing quinidine indefinitely, unless the patient got frequent recurrences. The tolerance for quinidine varied greatly in different individuals. A man working in a country goods shed had been six months away from work on account of auricular fibrillation. This had been stopped by two doses of quinidine and he had since been working for years with no relapses.

Gradually advancing organic disease frequently caused little distress which depended rather on the acuteness of onset. A girl with a 13·5 kilogram (thirty pound) ovarian cyst had been able to play tennis without much distress and a medical student with an extensive pleural effusion had had only slight dyspnœa after a game. Slow closure of a coronary arterial branch might thus not be associated with angina.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Pye, Aubrey Daird Dick, M.B., Ch.M., 1925 (Univ. Sydney), Kurrajong.

Medical Societies.

SAINT VINCENT'S HOSPITAL CLINICAL SOCIETY.

A MEETING OF SAINT VINCENT'S HOSPITAL CLINICAL SOCIETY was held at Saint Vincent's Hospital, Melbourne, on May 23, 1928, MR. GORDON SHAW, the President, in the chair.

Malignant Disease of the Liver.

MR. FORBES MACKENZIE showed a female patient, aged fifty-six years, who had spent all her life in the country. She presented a very great hepatic enlargement, mostly in the forward and downward directions. X ray examination with the screen showed that there was no backward enlargement and the base of the right lung was resonant to percussion. A large accessory lobe could be felt with what was thought to be nodulation. The only history was of pain in the right upper quadrant for the previous three weeks; her only previous illness was pleurisy and she had not lost much weight. The Casoni test yielded a strongly positive result.

Mr. Mackenzie thought the condition one of malignant disease of the liver and expressed his disappointment with the results of the Casoni test; quite a number of patients with uncomplicated hydatid cysts on which he had operated, had given no reaction. He proposed to explore the abdomen.

DR. A. J. BRENAN thought the Casoni test very reliable.

Diarrhœa of Obscure Origin.

Mr. Forbes Mackenzie's second patient was a male, aged twenty-six years, who had suffered from diarrhœa all his life. The stools were large, contained some mucus and often some undigested food. His general condition was good, there being very little evidence of dehydration. All the usual bacteriological, X ray and sigmoidoscopic examinations had revealed nothing but a slight enteroptosis. At rest under observation his stools were somewhat fewer. Mr. Mackenzie had explored the abdomen and found nothing but a funnel-shaped caecum and appendix which he removed. The patient had improved for a time, but was now as bad as ever. The condition somewhat resembled sprue of which he had had some experience in India. He asked for expressions of opinion.

DR. SYDNEY PERN cited cases similar to this in which the condition had cleared up on removal of septic foci (teeth, tonsils, sinuses) and suggested a careful investigation of these sites in the patient presented.

Spinal Lesion.

MR. H. S. BUSH showed a male, aged eighteen years, with a poker back and synovitis of the left knee. For seven years he had had pains in the back and lower limbs. As a child he had had pains in the hips and had been in bed for six months with a diagnosis of tuberculosis of the hip. One year previously he had been put to bed for six months with a tentative diagnosis of tuberculosis of the spine. Since his appearance at Saint Vincent's Hospital X rays had consistently failed to demonstrate any definite bony or ligamentous change, either of the spine or lower limbs. There had never been any pain or stiffness in the upper limbs or temporo-maxillary joint and the examination of the central nervous system revealed no abnormality.

MR. E. A. SPOWERS thought the condition was probably infective in origin, but suggested injecting a guinea pig with some of the synovial fluid.

MR. FORBES MACKENZIE thought the synovitis was probably due to the efforts of the boy to walk on a limb the

muscles of which were in such poor condition. He thought the condition was probably tuberculous with some pressure on the cord.

MR. CHARLES OSBORNE considered the evidence of a tuberculous nature in this case to be negligible and suggested as possibilities an infective focus, Neisserian infection, neuromymesis.

Fracture of Hyoid Bone.

Mr. Bush also showed a female, aged fifty-one years, who had undergone partial thyreoidectomy fifteen years and again twelve years previously. A small lump had persisted in the mid-line of the neck ever since. Following a recent altercation in which she had been grasped roughly by the neck, the lump had increased rapidly in size; it was about the size of an egg, non-translucent and fluctuant. On palpation of the hyoid bone, definite crepitus could be felt. The remarkable feature of the tumour was that it would suddenly subside, at which times the patient was conscious of an accumulation of fluid in the mouth. X ray examination revealed a fracture at the junction of the body and greater cornu of the hyoid.

MR. CHARLES OSBORNE thought the tumour was probably a hæmatoma, but the question of a thyreo-glossal cyst had to be considered.

Bronchiectasis and Hyperthyreoidism.

DR. L. S. LATHAM showed a female patient, aged fifty-eight years, with constant cough and copious sputum in which no tubercle bacilli could be found, although repeated examinations had been made. X ray examinations revealed a widespread pulmonary fibrosis and "Lipiodol" injection displayed cylindrical bronchiectasis at the right base. This patient also had definite symptoms of hyperthyreoidism, a pulse rate of 102, tremor, moist skin and a basal metabolic rate of +40%. There was no enlargement of the thyreoid.

Dr. Latham did not think surgical interference was warranted in this case, as the patient was making very good progress under medical treatment.

Spastic Paraplegia.

Dr. Latham also showed a female patient, aged sixty-five years, with spastic paraplegia, gradually progressive over a period of eighteen months. In the area corresponding with the distribution of the eleventh thoracic segment there was a sensation of a tight band and definite hyperæsthesia. Otherwise there were no subjective sensory phenomena. The reflexes in the upper extremity were very slightly increased, but beyond this there was nothing objective above the level of the eleventh thoracic segment. Intrathecal "Lipiodol" injection had failed to demonstrate a focal lesion.

Dr. Latham thought that there was probably a tumour in the region of the eleventh thoracic segment, the increase in arm reflexes being explained on a functional basis. In view of the age of the patient and of her comparatively comfortable state, operation was not recommended.

The question of Landry's paralysis and syringomyelia was discussed.

Calcified Hydatid Cysts.

DR. J. W. GRIEVE showed a female patient, aged fifty-six years, with a three years' history of recurrent left basal pleurisy, lethargy and loss of weight. X ray examination revealed an old calcified hydatid cyst about fifteen centimetres (six inches) in diameter in the left hypochondrium. It was remarkable that no mass could be felt. The result of the Casoni test was strongly positive. There were several small calcified areas in the liver also. The cyst had produced gross displacement of the stomach.

DR. A. J. BRENAN (for Dr. Colquhoun) showed a man with a very large primary syphilitic sore on the lip and secondary rash well developed. The sore had followed a bite.

MR. FORBES MACKENZIE in discussing the treatment of syphilis said that he thought that the old intramuscular injection of "606" gave better results than the more recent intravenous methods, but was more painful.

DR. E. A. SPOWERS thought bismuth should be given a more extensive trial. He referred to the value of grey powder in the treatment of syphilis in pregnant women whose pregnancy failed to reach full time. He got good results in several instances, even though the result of the Wassermann test was still positive at the end of treatment. He strongly urged the continued use of grey powder throughout the whole period of pregnancy.

Dr. A. J. Brenan in reply agreed with the French teaching that prolonged treatment was necessary, repeated absence of reaction to the Wassermann test being required as a standard of cure. Milian regarded bismuth as an adjuvant to "Salvarsan" and was not to replace it in the treatment of syphilis.

Obituary.

WILLIAM ALEXANDER MORTON.

DR. WILLIAM ALEXANDER MORTON died on May 9, 1928, aged fifty-three years, at Sunbury, Victoria, where he had practised since his return from the war. He was the son of the late David Murray Morton and brother of Dr. J. C. Morton, of Seymour, and Dr. D. Murray Morton, of Melbourne.

William Alexander Morton was educated at Christian Brothers' College, East Melbourne, and entered the Melbourne University in 1890. Here he excelled at sport, being a good footballer and runner. He graduated in 1895 and commenced practice in Outtrim, Victoria. He later moved to Merino, where he practised for about twenty years. On the outbreak of war he relinquished his practice and joined the Australian Imperial Force as a Captain in the Australian Army Medical Corps. He was abroad for four years and was for three years Medical Officer to the Third Field Artillery Brigade.

His military service was a record of great gallantry and devotion. He refused promotion in order to remain with his brigade and few, if any, Australian medical officers had longer continuous service in the field. He was promoted to the rank of major and was awarded the Distinguished Service Order; he was three times mentioned in dispatches. He was gassed and this, together with other hardships in the field, affected his health and indirectly hastened his death.

As a practitioner he was sound and careful. His kindness and unselfishness made him an ideal country doctor and he was much beloved by his patients.

His war service was not necessary to prove his courage and self sacrifice, for when as a younger man at Outtrim he was riding on horseback past a cottage, he was stopped and told that a child had fallen down a well. He immediately dismounted, stripped and dived into the well. After some difficulty he recovered the child, brought it to the surface and followed up his efforts by applying artificial respiration which after a long time was successful, and the child recovered. This incident was recognized by the presentation to him of an illuminated address by the people of Outtrim.

Such a man was William Alexander Morton, quite uncommercial, and leaving behind him little of worldly goods, but much of love and gratitude and an example which was a credit to his profession and his country.

Correspondence.

NURSES AND OVERWORK.

SIR: May I draw your attention to the need for nurses attending cases in private houses to have a sufficiency of time off duty for rest and recreation.

The general rule laid down by my Council has been eight hours' sleep and two hours at least for exercise daily. It is recognized, of course, that in the early stages of acute illnesses it is not possible to adhere strictly to these hours. In cases, however, where much duty is

necessary and where proper attention to the patient would entail broken rest over a period of more than three or four days, it should be recognized that either there should be two nurses engaged or a member of the household should attend to the more simple wants of the patient, that is, routine administration of mixtures *et cetera* during either a night or day period, so that the nurse may have a definite time for sleep. So often one finds that after a difficult case it is necessary for the nurse to have a week's rest before taking a fresh case. In this way it can be readily seen that either the earning capacity of the nurse is lessened by periods of enforced idleness without pay or her health suffers.

It was decided to write to you to ask your cooperation by publishing this letter, as complaints about the amount of duty to be done by nurses are almost always referred to the medical man in charge of the case and, while he cannot lightly impose extra financial burdens on his patients, he still must protect the nurses from overwork.

Yours, etc.,
E. P. EVANS,
Secretary.
Australian Nursing Federation.

185, Elizabeth Street, Sydney.
June 2, 1928.

DIPHTHERIA INFECTION CONVEYED BY MILK.

SIR: That milk is capable of being the vehicle for the carriage of diphtheria infection is, of course, acknowledged, but the Australian records on the subject are meagre.

The following example is of interest and doubtless if investigations were made in a routine manner into every case that was notified, milk would be found to be a greater disseminator of diphtheria than is supposed at present.

Between April 2 and May 4, nineteen cases of diphtheria were reported and five carriers detected, all of the twenty-four persons concerned living within a fairly circumscribed area. On investigation it was found that all of these households were served by the same milk vendor who obtained his supplies from a dairy some miles distant. The occupants and workers at the dairy were swabbed, but no carriers were found; the cows also were inspected with negative results. The milk vendor's household and staff were then examined and a man in charge of a milk cart was found to be a virulent carrier. This man was immediately removed from his work and isolated. From the date of his admission to hospital no further cases of diphtheria occurred in that particular locality or amongst any of the customers of that vendor.

Whilst it is impossible to state definitely the way in which the milk became infected, in all probability it was effected by either the cans or measures being contaminated by personal contact or by an infected cloth.

Yours, etc.,
H. W. TILLING,
Medical Officer of Health,
City of Brisbane.

Brisbane.
June 8, 1928.

FALLOPIAN TUBE IN A FEMORAL HERNIA.

SIR: The following description of a case of femoral hernia may be of sufficient interest for publication.

Mrs. E., aged twenty-eight, on June 1 felt pain and noticed swelling in the left groin. She was accustomed to help her husband on his farm and to lift weights such as cream cans. The pain and swelling continued and became worse, so that she consulted me on June 3. She had not vomited and the bowels had acted naturally. The swelling was the size of a hen's egg over the saphenous opening, not tympanitic and not unduly tender. It could not be replaced. I thought that some omentum only was incarcerated as the symptoms discounted the presence of bowel. At operation next day the extraperitoneal fat was necrotic and showed blood clot in patches. The sac was distended with clear fluid and on opening this the congested and swollen left Fallopian tube presented. It was very tightly constricted and only after deeply cutting Gimbernat's ligament could be replaced. The patient recovered.

Yours, etc.,
E. R. ROSEBY, M.B., Ch.M.

Dorrigo, New South Wales.
June 11, 1928.

Medical Prizes.

PRIZE ESSAY.

THE Council of the British Medical Association proposes to award a prize of £25 for the best essay on the symptoms and sequelæ of *encephalitis lethargica*, with their appropriate treatment, by a medical student in the final year of any medical school in the British Empire outside the British Isles. The award will be made in March, 1929. No prize will be granted if none of the essays submitted is deemed to be deserving of a prize. The essays must not exceed five thousand words, must be clinical in nature and must contain concise notes of three cases personally studied by the essayist. The essays must be plainly written or typed on foolscap paper on one side. They must reach the Medical Secretary not later than January 12, 1929. Each essay must be signed by a pseudonym and be accompanied by a signed and dated statement that it has been the *bona fide* work of the competitor and that he or she had not yet passed the final professional examination, together with particulars of his or her name, including full Christian name or names, the pseudonym used, the address and the medical school. The address of the Medical Secretary is British Medical Association House, Tavistock Square, London, W.C.1.

Medical Research.

RESEARCH IN OPHTHALMOLOGY.

AN announcement is made that the editorial committee of *The British Journal of Ophthalmology* is prepared to assist research work in ophthalmology by granting subsidies towards the expenses of the research to those qualified to undertake such work. The qualifications of the applicant, the subject of the proposed investigations and the place where the work is to be carried out must be submitted to the editorial committee. An estimate of the probable time and cost necessary to complete the work must be given and the amount of financial assistance required must be indicated. The committee reserves to itself the right to publish in *The British Journal of Ophthalmology* any paper written on the results of the assisted investigations. All applications should be addressed to the editor of *The British Journal of Ophthalmology*, 24, Thayer Street, Manchester Square, London, W.1.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of *The Medical Act* of 1915, of Queensland, as duly qualified medical practitioners:

Lynch, John Adrian, M.B., B.S., 1925 (Univ. Melbourne), Brisbane.

Henderson, John Hector Baillie, M.B., B.S., 1928 (Univ. Sydney), Brisbane.

Murphy, Hugh Clement, M.B., B.S., 1928 (Univ. Sydney), Brisbane.

Spark, Robin James Hester, M.B., Ch.M., 1926 (Univ. Sydney), Gatton.
Fryberg, Abraham, M.B., B.S., 1928 (Univ. Melbourne), Brisbane.
Biggs, Thomas Roy, M.B., B.S., 1928 (Univ. Sydney), Brisbane.
Joyce, John King, M.B., B.S., 1928 (Univ. Melbourne), Ipswich.
Little, Leonard Andrew, M.B., B.S., 1928 (Univ. Melbourne), Brisbane.

Restorations to the Register:

Berry, Robert Sewers, L.S.A. (London), 1892, M.R.C.S. (England), 1893, L.R.C.P. (London), 1893, Southport.
Jackson, John Henry Sandford, M.B., B.S., 1919 (Univ. Melbourne), Baralaba.

Books Received.

SYSTEMIC INFECTIONS: THEIR DIAGNOSIS AND TREATMENT, by A. Knyvett Gordon, M.B., B.C., B.A. (Cantab.); 1928. London: Baillière, Tindall and Cox. Demy 8vo., pp. 186. Price: 10s. 6d. net.

THE HEALERS, by B. Liber; 1928. New York: Rational Living. Crown 8vo., pp. 454. Price: $3.00 net.

Diary for the Month.

JUNE 22.—Queensland Branch, B.M.A.: Council.
JUNE 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 27.—Victorian Branch, B.M.A.: Council.
JUNE 28.—New South Wales Branch, B.M.A.: Branch.
JUNE 28.—South Australian Branch, B.M.A.: Branch.
JUNE 30.—Eastern District Medical Association, New South Wales.
JULY 3.—Tasmanian Branch, B.M.A.: Council.
JULY 3.—New South Wales Branch, B.M.A.: Council (Quarterly).
JULY 4.—Victorian Branch, B.M.A.: Branch.
JULY 4.—Western Australian Branch, B.M.A.: Council.
JULY 5.—South Australian Branch, B.M.A.: Council.
JULY 6.—Queensland Branch, B.M.A.: Branch.
JULY 10.—Tasmanian Branch, B.M.A.: Branch.
JULY 10.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 12.—Victorian Branch, B.M.A.: Council.

Medical Appointments.

Dr. John Coffey (B.M.A.) has been appointed Deputy Commissioner of Public Health and Deputy Inspector, School of Anatomy, Queensland.

* * * *

Dr. Peter McLean Davidson (B.M.A.) has been appointed Government Medical Officer at Proserpine, Queensland.

* * * *

Dr. R. H. Minton Connell has been appointed Medical Officer of Health for the Portland Municipality, Tasmania.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi..

AUGATHELLA HOSPITAL, QUEENSLAND: Medical Superintendent.
AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Radiologist, Pathologist and Registrar.
LAUNCESTON PUBLIC HOSPITAL: Junior Resident Medical Officer (male).
THE UNIVERSITY OF MELBOURNE: Stewart Lectureship in Pathology.
WESTERN AUSTRALIAN STATE PUBLIC SERVICE: Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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